Yayouk Eva Willems

Out of Control: Causes of Individual Differences in Self-Control

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Out of Control: Causes of Individual Differences in Self-Control

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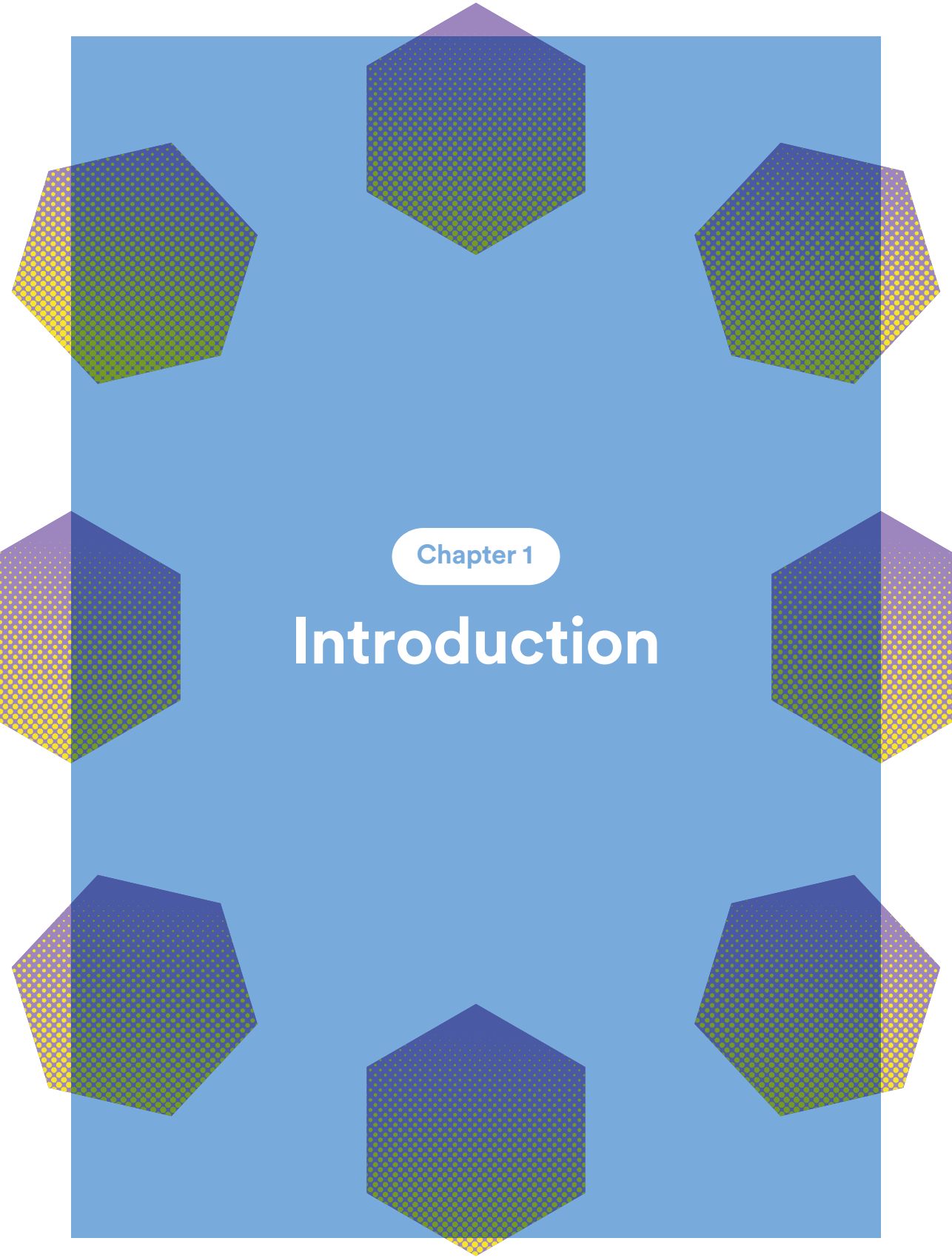
Chapter 1
Introduction

Life is marked by a range of self-control challenges. Individuals have to focus on their (school) work while being distracted by social media, control their insecurities when forming new relationships, and regulate their food intake while tempted by sugar coated goodies. Failing to exert self-control places individuals at risk for myriad psychological and physiological problems. For example, individuals with low self-control have poorer career prospects, display unhealthier lifestyles (e.g. less exercise, more obesity, more alcohol intake), and are more likely to experience mental health problems than individuals with high self-control (Caspi et al., 2016; de Ridder, Lensvelt-Mulders, Finkenauer, Stok, & Baumeister, 2012; Moffitt et al., 2011). It is therefore important to **identify factors that shape self-control throughout the life course.**

A multitude of factors potentially shape the way we are, ranging from factors on a micro level (e.g. genes) to factors on a macro level (e.g. society). Investigating these factors is complex, as they are nested in individuals and contexts, which also reciprocally influence one another (Bronfenbrenner, 1979). Traditionally, much research has focused on contextual influences, investigating the way in which family factors influence individual differences. In the last decades, with the accumulation of twin data and the affordability of molecular genetic techniques, a growing line of research has examined genetic sources of individual differences. Consequently, while in the past the **"Nature versus Nurture"** debate dominated the discourse, currently there is increasing awareness that "**Nature and Nurture"** jointly explain how individual differences in the population arise (Harold, Leve, & Sellers, 2017; Plomin, DeFries, Knopik, & Neiderhiser, 2016).

Useful concepts to understand the interplay between environmental and genetic factors are **gene-environment correlations** (rGE) and **gene by environment interactions** (G x E). rGE describes the process by which someone's genotype correlates with his or her environmental exposure (Kendler & Eaves, 1986; Plomin, DeFries, & Loehlin, 1977). For example, the observed correlation between growing up in a household full of books and being good at reading is not necessarily causal as it can be (partly) explained by parents transmitting the genetic propensity to be good at reading and providing an environment where a child is stimulated to read. When investigating causality, it is therefore important to take into account gene-environment correlation as it possibly confounds the relationship between exposure (the number of books) and the outcome (being good at reading, D'Onofrio, Lahey, Turkheimer, & Lichtenstein, 2013; Pingault et al., 2018). G x E describes the process where certain genotypes vary in their sensitivity to certain environments. For example, it is

hypothesized that people can experience a similar life stressors but those with a genetic vulnerability are more likely to develop depression as a result of this environmental exposure than those with a lesser genetic vulnerability (Monroe & Simons, 1991).

While these are interesting concepts, they are **difficult to empirically test**: One needs data integrating both environmental and genetic information, have the appropriate study design, that allow one to examine the gene-environment interplay, and have the statistical power to detect an hypothesized effect (Jaffee, 2016). While there have been increasing efforts to meet these conditions (including the gathering of genetically sensitive data, collaboration between multidisciplinary research groups, and development of cutting-edge statistical models, Boomsma, Busjahn, & Peltonen, 2002), thus far few studies looked at the dynamic interplay between genes and the environment for self-control specifically. However, doing so is important if we truly want to understand how individual differences in self-control in the population arise. The aim of this dissertation is therefore two-fold. First, **we take stock of the literature** by systematically reviewing to what extent environmental and genetic influences explain individual differences in self-control. Second, **we investigate the etiology of self-control taking gene-environment interplay into account** by using a wide variety of genetically-sensitive research designs. In this introduction, we discuss the definition of self-control, its importance for psychological and physiological well-being, and the way in which environmental and genetic factors possibly shape self-control during childhood and adolescence.

Conceptualizing Self-Control: It's Complicated

Self-control is the ability to alter unwanted impulses and behaviors in order to bring them into agreement with goal-driven responses (Duckworth & Steinberg, 2015; Tangney, Baumeister, & Boone, 2004). Considering the robust associations between self-control capacities and psychological and physiological health, self-control is widely studied in the (social) sciences. The nomenclature of self-control capacities, however, widely varies across disciplines. For example, while clinicians and developmental psychologists often refer to **self-regulation** (Bridgett, Burt, Edwards, & Deater-Deckard, 2015), **self-control** is more commonly used in health psychology, social psychology, and criminology (Gottfredson & Hirschi, 1990; Tangney et al., 2004), **executive functioning** and **grit** are more frequently used in the educational and cognitive sciences (Diamond, 2013; Duckworth, Peterson, Matthews, &

Kelly, 2007), and temperament and personality researchers more often refer to **effortful control** and **conscientiousness** (Funder, 2001; Rothbart, Ellis, Rueda, & Posner, 2003). Not surprisingly, the multitude of these concepts from different disciplines, with a focus on specific self-regulating aspects or developmental life stages, resulted in considerable confusion and boisterous debates concerning their similarities and differences.

While this conceptual confusion is far from resolved, the number of unifying **reviews comparing and contrasting self-control related concepts** is increasing (e.g. de Ridder, Lensvelt-Mulders, Finkenauer, Stok, & Baumeister, 2012; Diamond, 2013; Nigg, 2017; Zhou, Chen, & Main, 2012). A useful framework repeatedly put forward in these reviews to distinguish self-control related concepts is the "dual-process framework" (Evans, 2008; Hofmann, Friese, & Strack, 2009; Metcalfe & Mischel, 1999). This framework distinguishes top-down and bottom-up regulation processes, commonly juxtaposed as, for example, regulated/regulating, type II/type I, conscious/unconscious, deliberate/automatic, explicit/implicit, or endogenous/exogenous. Top-down processes refer to conscious and deliberate efforts, stimulating desirable responses over undesirable impulses while reflecting internal mental representations and goals (Nigg, 2017). For example, they entail the deliberate regulatory process where one has to consciously choose between a reward now or gratification later. Bottom-up regulation processes refer to automatic, stimulus driven impulses elicited by external or internal stimuli (Nigg, 2017). For instance, the startling response to a loud noise, the anxious feeling when seeing a spider, or waking up when having a full bladder. While often described as opposing categories, top-down and bottom-up processes are better understood as different aspects of a continuum. They are connected to one-another through reciprocal interactions: Top-down processes can control or activate bottom-up processes, and bottom-up processes can elicit or limit goal-directed top-down processes (Evans & Stanovich, 2013). For example, bottom-up systems influence top-down processes through priming or emotional perceptual properties and top-down processes can suppress or bias bottom-up responses (Nigg, 2017).

Generally, **self-regulation** encompasses both top-down and bottom-up regulating processes. Consequently, self-regulation is considered as an overarching concept (an 'umbrella term'), under which other (more specific) self-regulating concepts can be placed, such as self-control (Kelley, Wagner, & Heatherton, 2015). **Self-control** entails top-down efforts to alter thoughts, emotions, and behaviors, and bring them in agreement to reach long-term goals (Duckworth & Steinberg, 2015). For instance, a PhD student faces

conflict when choosing between finishing the dissertation introduction and scrolling through fashion photos on Instagram. Scrolling through Instagram is satisfying and fun in the short run, but not useful in the long run. On the contrary, finishing the dissertation introduction is not satisfying in the moment but useful on the long run. The student cannot do both at once, so the student needs to perform self-control: Exerting conscious regulating of thoughts and emotions not to check Instagram, and align behavior with the long-term goal to acquire that long desired PhD-degree. Considering the involvement of self-governing and conscious processes, many theorists consider self-control as the top-down aspect of self-regulation (de Ridder et al., 2012; Finkenauer, Engels, & Baumeister, 2005).

Two additional concepts that encompass top-down processes are executive functioning and grit. **Executive functioning** is conceptualized as the interrelated cognitive operations involved in the top-down control of thoughts, emotions, and behaviors (Diamond, 2013). These functions develop hierarchically with age, starting in early childhood with 'low level executive functioning' (attention shifting, working memory), and later developing into 'high level executive functioning' (planning, problem-solving, Miyake et al., 2000; Zelazo & Carlson, 2012). Executive functions are necessary for top-down self-control processes to occur (e.g. planning, attentional control, inhibitory control), and therefore some consider executive functioning and self-control to closely overlap (Hofmann, Schmeichel, & Baddeley, 2012). Others, however, argue that executive functioning and self-control cannot simply be equated, as executive functioning comprises merely the 'cognitive tools' for self-control to occur, with executive functioning being a facet of an overarching self-control process (Baumeister, 2002). While the concept 'executive functioning' has been the focus of scientific research for a long time, more recently a new concept entered the 'top-down processing realm', namely grit. **Grit** is conceptualized as the passion and effort to reach long-term goals, and the capacity to persist despite obstacles and failures (Duckworth et al., 2007). As such, some consider grit to be distinct from self-control as it more specifically focusses on long-term endeavors incorporating perseverance and motivation (Duckworth & Gross, 2014). Others, however, are more skeptical concerning the uniqueness of grit, arguing grit is just a different "label" for self-control (Credé, Tynan, & Harms, 2017).

While the previously mentioned self-control related concepts do not necessarily focus on a specific age group, the concept **effortful control** typically concerns (early)childhood. Effortful control emanates from the temperament

literature, traditionally focusing on top-down regulating dispositional traits early in life, representing individual differences in the reactivity and regulation of emotions, activity and attention (Kochanska, Murray, & Harlan, 2000; Rothbart et al., 2003). Especially in children, effortful control and self-control are considered to be very similar as effortful control is conceptualized as the voluntary efforts undertaken to manage attention, influence feeling stages, and to activate adaptive behavior (Kochanska et al., 2000). Others, however, argue self-control to be broader than effortful control, considering its emphasis on advancing distal goals over proximal impulses (Fujita, 2011).

Moving from childhood to adulthood, temperament is believed to underlie the emergence of later personality traits, with effortful control in childhood as a likely precursor of conscientiousness in adulthood (Eisenberg, Duckworth, Spinrad, & Valiente, 2014). **Conscientiousness** originates from the personality literature, and typically concerns the set of cognitions, beliefs, emotional patterns, and behaviors in adulthood including both top-down and bottom-up regulating processes. Conscientiousness is a personality trait that is defined as the propensity to show self-discipline, follow norms, and successfully delay gratification (Funder, 2001). Taking this broad-definition, some argue that self-control, executive functioning, and grit are separable facets of the overarching conscientiousness construct (Nigg, 2017). Others, however, argue self-control is not an element of conscientiousness, but that people with high self-control are likely to have a profile that combines different personality dimensions, such as low neuroticism, high conscientiousness, and high agreeableness (Sharma, Markon, & Clark, 2014).

From a theoretical standpoint, there is a fine line between the similarities and differences across these different self-control related capacities. From a practical standpoint, this line becomes thinner because **empirical consensus is lacking on whether and to what extent these concepts statistically tap into unique or common capacities**. On the one hand, research emphasizes these self-control related concepts cannot simply be equated. For example, grit researchers repeatedly emphasize the unique contribution of grit to academic success over and above self-control (Duckworth & Gross, 2014). Additionally, correlations between self-reported self-control and executive functioning measures (e.g. Stroop test, Flanker task) are small to zero (Duckworth & Kern, 2011; Saunders, Milyavskaya, Etz, Randles, & Inzlicht, 2018). This lack of correlation does not invalidate either of these measures, yet it emphasizes that we have to rethink the convergent validity amongst measures and the way these measures differentially tap into self-regulating capacities (Malanchini,

Engelhardt, Grotzinger, Harden & Tucker-Drob, 2018). On the other hand, a growing line of research emphasizes the strong overlap among these self-control related capacities. For example, self-reports of self-regulation, self-control, effortful control, grit, and conscientiousness correlate around .70 or higher (Credé et al., 2017; Duckworth & Kern, 2011). Additionally, commonality analyses suggest that it is the overlap between these traits that accounts for the explained variance in health-related outcomes, rather than the individual contributions of these separate concepts (Werner, Milyavskaya, Klimo, & Levine, 2019).

Although many scientists study self-control or related concepts, questions surrounding the commonalities and differences between self-control related concepts remain to be answered. Nevertheless, overall the literature converges to suggest that self-control and related concepts tap into the capacity to override undesirable impulses, thoughts, and emotions and align behaviors with valued, long term goals. Therefore, instead of focusing on the differences among self-control and related capacities, **in this dissertation, we use the term self-control referring to this commonality between concepts**, while recognizing its multidisciplinary nature and close relation to related concepts such as self-regulation, executive functioning, grit, effortful control, and conscientiousness.

Why Focus on Self-Control?

The famous "marshmallow experiment" by Walter Mischel and his colleagues (1989) is a well-known self-control test inside and outside academia. In this experiment, children around the age of four get one marshmallow and are offered a second one if they are able to wait for 15 minutes. It gained fame not only because of its online footage (7,5 million views on YouTube), but also because of the interesting study outcomes; children who were not able to wait for the second marshmallow showed worse school performance and less social competence 10 years later as compared to children who were able to wait for the second marshmallow (Mischel, Shoda, & Rodriguez, 1989). While the effect sizes of this specific experiment are currently debated (Watts, Duncan, & Quan, 2018), a wide variety of longitudinal studies show that **low self-control in childhood is predictive of myriad negative outcomes**. For example, low self-control during childhood is predictive of worse physical health, lower school performance and personal financial issues, and more substance dependence and criminal offending later in life (Caspi et al., 2016; Duckworth, Tsukayama, & Kirby, 2013; Moffitt et al., 2011; Tsukayama, Toomey, Faith, & Duckworth,

2010). Thus, the level of self-control children have can shape how well they do in school, relationships, work and health related behaviors throughout the lifespan.

The capacity to exert **self-control is of specific importance to adolescents**. The teenage years (10-24 years, Sawyer, Azzopardi, Wickremarathne, & Patton, 2018) are marked by a range of biological and social challenges: Adolescents face a multitude of bodily and cognitive changes, have to form new identities when transitioning to high-school, conform to parental rules while striving for independence, and regulate emotions and insecurities in online and offline relationships (Casey & Caudle, 2013; Crone & Dahl, 2012a; Meeus, Van De Schoot, Keijsers, Schwartz, & Branje, 2010). Adolescence is also a period featured by increased risk taking and social reward seeking, coining adolescence as a period that has lasting implications for youth's trajectories of economic security, health, and well-being into later life (Patton et al., 2016; Harden & Tucker-Drob, 2011). Self-control aids adolescents in dealing with these challenges and provides them with the tools and capacities necessary to regulate their thoughts, emotions, and impulses and align their behaviors with long-term goals. In this dissertation, we therefore particularly focus on factors shaping self-control throughout adolescence.

Environmental and Genetic Influences on Self-Control

Traditionally, environmental and genetic influences on human behavior were studied in distinct disciplines (cf. environmentalists versus geneticists). However, environmental factors like parenting containing genetic aspects and genetic variants can only be expressed in certain environments. While we are aware it is the interplay between environmental and genetic influences that explain differences in human behavior, **we start by summarizing the literature** of the two traditionally different lines of research.

An extensive line of work in early and middle childhood illustrates associations between **parenting and self-control** (Bridgett, Burt, Edwards, & Deater-Deckard, 2015; Kochanska et al., 2000). Socialization theories highlight that parents help children to develop their self-control skills (Kopp, 1992; Sameroff, 2010; Sroufe, 1996). On the one hand, parents who employ positive parenting strategies (e.g. warmth, monitoring, family connectedness) create a secure and safe context where children learn to regulate their inner feelings and impulses. In such a safe environment, children can work through problems themselves thereby developing autonomy and internalizing rules and expectations of appropriate behaviors. On the other hand, parents who

employ negative parenting strategies jeopardize children's ability to develop self-control. For example, hostile parenting practices cumulate stressful family contexts hampering children to learn how to regulate their emotions and impulses, and over controlling parenting practices do not grant children the autonomy to learn how to self-regulate or to set autonomous goals for the future (Srouffe, 1996). As such, positive parenting strategies have generally been positively associated with self-control while negative parenting strategies have been negatively associated with self-control development across early and middle childhood (Karreman, van Tuijl, van Aken, & Deković, 2006; Pallini et al., 2018).

However, studies extending this work to adolescence yield mixed findings. Adolescence is a distinct and transitional phase in life. Adolescents spend less time in the household, demand more independence and rely more on their friends and less on their parents than children (Patton et al., 2016). This raises the important question: Is parenting still associated with self-control across adolescence? Additionally, children and adolescents are active agents in their own development, and the way they behave also steers the way their parents parent. For example, an adolescent who is not meeting the Saturday night's curfew multiple times in a row is more likely to evoke more controlling parenting than an adolescent who is always home on time (Sameroff, 2010). This raises the question: to what extent does adolescent self-control influence parenting practices? In Chapter 2, we used a meta-analytic approach to find answers to these questions, **quantifying the overall relationship and the direction of the relationship between parenting and self-control across adolescence**. Synthesizing the results of the multitude of published studies allowed us to systematically summarize the research thus far, quantifying the overall association between parenting and self-control, and gain insight into possible moderators influencing the magnitude of this association.

More recently, theoretical work specifically points to the association between **family violence and self-control** (Finkenauer, Büyükcan, Schoemaker, Willems, Bartels, & Baumeister, 2018; Finkenauer et al., 2015). Family violence and its coinciding stressors is assumed to negatively influence self-control development (Davies & Cummings, 1994), depleting emotional and psychological resources necessary for self-control exertion (Baumeister, Vohs, & Tice, 2007), and/or provide an environment that is not conducive to exert self-control (Ellis, Bianchi, Griskevicius, & Frankenhuis, 2017). However, an examination of the strength of the relationship between family violence and self-control is lacking. In Chapter 3, we therefore conducted a **meta-analysis**

quantifying the overall relationship between family violence and self-control from early adolescence to early adulthood. Doing so allowed us to summarize the associations found in the published literature thus far, and to explore under what conditions the magnitude of this association changes. For example, is the magnitude of the association equal in early and late adolescence? Is the association similar for boys and girls? And to what extent do methodological factors influence the association? By applying moderator analyses, we aimed to provide answers to such questions.

A different line of research, originating from **behavioral genetics**, postulates that traits like self-control are also influenced by biological factors including genes (Polderman et al., 2015). The overall influence of genes is frequently expressed as 'heritability', with an heritability estimate reflecting the extent to which individual differences between people on a certain trait are explained by genetic differences between people (Boomsma et al., 2002). Thus far, heritability estimates for self-control range between 0% (differences in people's self-control are not explained at all by genetic differences between people) and 90% (differences in people's self-control are almost completely explained by genetic differences between people), with a large variety in the quality of studies, mostly due to variance in sample sizes (Beaver, Connolly, Schwartz, Al-Ghamdi, & Kobeisy, 2013; Bridgett, Burt, Edwards, & Deater-Deckard, 2015; Gagne & Saudino, 2016). To gain insight into the extent to which individual differences in self-control are explained by environmental and genetic factors, in Chapter 4 we conducted a **meta-analysis synthesizing studies reporting on heritability estimates of self-control**. By taking stock of genetic studies published thus far, we can paint a more complete picture of the heritability of self-control and the effect of environmental factors.

Causes of Individual Differences in Self-Control

The meta-analyses allowed us to provide a state-of-the-art overview of the current literature on the extent that environmental factors (parenting, family violence) and genetic factors (heritability) are related to self-control. While these studies quantify the overall importance of environmental and genetic factors for self-control, they do not reveal underlying mechanisms, including the causal role of the shaping factors, or the way they interact. **Investigating these underlying mechanisms is key**, as without this understanding it is hard to develop, or improve, intervention strategies for children and adolescents with low self-control. To bridge theoretical knowledge on contextual influences while using data with environmental and genetic information, we spearheaded

a collaboration between researchers from the Department of Interdisciplinary Social Sciences at Utrecht University and researchers from the Netherlands Twin Register (NTR).

The NTR is a large population-based twin and family study initiated in 1987 in the Netherlands at the Vrije Universiteit Amsterdam (VU), assessing the physical and psychological health of twins and their families from birth till adulthood (van Beijsterveldt et al., 2013; Willemsen et al., 2013). Data collected by this register allow researchers to apply various statistical models, such as the classical twin model. The classical twin model is built upon the premise that differences in the resemblance between monozygotic twins (sharing approximately 100% of their segregating genes) and dizygotic twins (sharing on average 50% of their segregating genes) can be used to parse phenotypic trait variance into genetic and environmental variance (Boomsma et al., 2002). This analysis can be extended by other statistical models, such as the direction of causality model and monozygotic twin difference models to explore not only sources of variance but also the directions of effect (Bartels, de Moor, van der Aa, Boomsma, & de Geus, 2012;de Moor, Boomsma, Stubbe, Willemsen, & de Geus, 2012; Duffy & Martin, 1994; Heath et al., 1993). Thereby, the available data from the NTR provided us with the opportunity to further unravel the underlying mechanisms explaining self-control differences across the lifespan.

While the NTR data collection focused on general psychological and physical health, there has been no specific focus on the assessment of self-control. We therefore aimed to develop a self-control scale based on the readily available data, using the richness of the longitudinal population-based database. In Chapter 5, we investigated whether a theoretically-derived set of items selected from the widely used Achenbach System of Empirically Based Assessment (ASEBA, Achenbach & Rescorla, 2001) can be used to assess self-control during childhood across different raters to form an **ASEBA Self-Control Scale**. Based on the literature, we selected 8 items, similar in content across age and rater, to establish a self-control scale and assessed its psychometric properties by examining internal and criterion validity, and inter-rater and test–retest reliability.

Next, we further investigated the processes studied in the meta-analyses by applying various twin models revealing underlying causalities and gene-environment interplay in the development of self-control. In Chapter 6, we examined the association between dimensions of positive family environment and self-control in 14- and 16-year-old twins. Here, we specifically focused on family connectedness – the feeling of trust, understanding, and support

within the family – which research has consistently associated with self-control development (Hagerty, Lynch-Sauer, Patusky, Bouwsema, & Collier, 1992; Pallini et al., 2018). We applied classical twin designs and monozygotic twin difference designs to reveal **to what extent the association between family connectedness and self-control is explained by environmental or genetic factors**, and whether the association is explained by causality or driven by possible confounding factors.

In Chapter 7, we aimed to investigate the association between negative family influences (family conflict) and self-control in adolescence. We applied direction of causality (DoC) models, using twin correlations to make predictions considering the direction of the effect (Duffy & Martin, 1994; Heath et al., 1993). Using data from 14-year-old twins, we tested whether the association between family conflict and self-control is best explained by a (1) reciprocal effect, (2) unidirectional effect from family conflict to low self-control, (3) unidirectional effect from low self-control to family conflict, or (4) a confounding factor such as genetic factors shared by parents and children. Doing so, allows us **to unravel what underlying mechanisms explain the association between family conflict and self-control in adolescence**.

Third, in Chapter 8 examined whether genetic risk and life stressors interact to predict self-control. In line with the G x E proposition (Kendler & Eaves, 1986; Plomin et al., 1977), we aimed to test whether the genetic propensity for low self-control interacts with experienced life stressors (e.g. violence, sexual abuse, losing a loved one), explaining why some have lower self-control than others in the population. Behavioral traits have a polygenic architecture, with traits being explained by a multitude of genes with a small magnitude across the genome rather than one gene explaining all the variance. This can be modeled by creating a polygenic score, which allows to calculate a genetic vulnerability score (a polygenic score, PS) resulting in a composite of additive effect of multiple genetic variants across the genome (Purcell et al., 2009). Taking this polygenic structure into account, we **tested whether genetic sensitivity (polygenic scores) interacts with environmental stressors (experienced life stress) as a shaping factor for self-control**.

In short, **in this dissertation we map factors that shape self-control**. We will do so by investigating factors on a micro level (the heritability in *Chapter 3*) to factors on a macro level (the family context in *Chapter 2* and *3*, and life stressors in *Chapter 8*). Using self-control data of a large sample of twins (*Chapter 5*), we aim to gain insight into the causality of these factors (*Chapter 6, 7* & *8*). Consequently, with this dissertation we investigate how both Nature and Nurture explain how self-control differences in the population arise.

Chapter 2

Parenting and self-control across early to late adolescence: A three-level meta-analysis

ABSTRACT

Self-control plays a significant role in positive youth development. Although numerous self-control challenges occur during adolescence, some adolescents control themselves better than others. Parenting is considered a critical factor that distinguishes adolescents with good self-control from those with poor self-control, but existing findings are inconsistent. This meta-analysis summarizes the overall relationship between parenting and self-control among adolescents aged 10 to 22. The analysis includes 191 articles reporting 1,540 effect sizes (N=164,459). The results show that parenting is associated with adolescents' self-control both concurrently (r=.204, p<.001) and longitudinally (r=.157, p<.001). Longitudinal studies also reveal that adolescents' self-control influences subsequent parenting (r=.155, p<.001). Moderator analyses show that the effect sizes are largely invariant across cultures, ethnicities, age of adolescents, and parent and youth gender. Our results point to the importance of parenting in individual differences in adolescent self-control and vice versa.

Keywords: parenting; parent-child relationship; self-control; adolescence; meta-analysis.

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Adolescence is a distinct and pivotal period of life with significant changes and growth at every level of functioning. What happens during adolescence has profound and long-lasting implications for youth's trajectories of economic security, health, well-being and development into later life (Patton et al., 2016). It is also a sensitive period featured by increasing prevalence of risk behaviors jeopardizing youth's physical and psychological functioning across their life course (Arnett, 1992, 1999; Casey, Jones, & Hare, 2008; Steinberg, 2004). Consequently, numerous studies investigated what factors counteract adolescents' problem behaviors and self-control is found to be such a factor (Moffit et al., 2011; Caspi et al., 2017). Self-control – defined as one's ability to alter dominant responses to abide by social values, moral norms, and to support the pursuit of long-term goals (Baumeister, Vohs, & Tice, 2007; Tangney, Baumeister, & Boone, 2004) – plays a key role in positive youth development and is widely studied across disciplines such as economics, public health and neuroscience (Caspi et al., 2017; Figner et al., 2010; Hare, Camerer, & Rangel, 2009). It helps adolescents get through a stage marked by a range of normative biological and social challenges (Crone & Dahl, 2012), increases in risk-taking and social reward seeking behavior (Boyer, 2006; Smetana, Campione-Barr, & Metzger, 2006), and heightened emotional turmoil (Steinberg & Morris, 2001).

To illustrate, adolescents with higher levels of self-control use less drugs and alcohol (Vazsonyi, Trejos-Castillo, & Huang, 2006), experience fewer emotional and behavioral problems (Finkenauer, Engels, & Baumeister, 2005; Li, Delvecchio, Lis, Nie, & Di Riso, 2015, Situ, Li, & Dou, 2016; Vazsonyi & Belliston, 2007), show more prosocial behavior (Nie, Li, & Vazsonyi, 2016; Padilla-Walker, & Christensen, 2011), and are more likely to achieve better academic accomplishments than adolescents with lower levels of self-control (Duckworth & Seligman, 2005; Galla & Duckworth, 2015). Self-control generally continues to improve throughout adolescence in terms of neural and psychological development (Casey, Jones, & Hare, 2008; Giedd, 2004; Romer, Duckworth, Sznitman, & Park, 2010), however, some adolescents show better self-control than others. To enhance our understanding of how these individual differences in self-control come about during the unique period of adolescence, the present meta-analysis sought to examine the relation between parenting and self-control across adolescence by considering the joint contribution of adolescents and parents to adolescent development.

Theorists agree that parenting is an important factor associated with individual differences in self-control (Bridgett, Burt, Edwards, & Deater-Deckard, 2015; Gottfredson & Hirschi, 1990; Kochanska, 1993; Kopp, 1982;

Sameroff, 2010). More specifically, prior studies found that positive parenting (e.g., monitoring, consistent discipline, parental warmth and support, positive control, authoritative parenting), and a strong parent-child relationship (e.g., secure attachment, close parent-child bonding, high quality of relationship) are related to better self-control. Conversely, negative parenting (e.g., inconsistent discipline, harsh parenting, coercive parenting, physical punishment, negative control, authoritarian parenting) and a weak parent-child relationship are associated with lower self-control in early and middle childhood (see meta-analyses: Davis, Bilms, & Suveg, 2017; Karreman, van Tuijl, van Aken, & Deković, 2006; Pallini et al., 2018). However, studies extending this work to adolescence yield mixed findings. While some studies report robust cross-sectional and longitudinal associations between parenting and self-control throughout adolescence (Hay, 2001; Hope et al., 2003; Özdemir et al., 2013; Vazsonyi & Belliston, 2007), others report only significant concurrent associations (Baardstu et al., 2017; Craig, 2015; Moilanen, Rasmussen, Padilla-Walker, 2014; Pallini et al., 2018), and some studies find significant associations for some parenting dimensions but not for others (Finkenauer et al., 2005; Vazsonyi et al., 2016). As such, previous findings regarding the magnitude and the direction of the association between parenting and self-control in adolescence are not conclusive.

These inconsistent findings might be explained by the notion that parenting is most strongly associated with self-control during early and middle childhood and less in other developmental periods (Kochanska, 1993; Gottfredson & Hirschi, 1990; Hay & Meldrum, 2016; Kopp, 1982; Meldrum, Young, Hay, & Flexon, 2012; Vazsonyi & Jiskrova, 2017). During early and middle childhood, children rely mostly on their parents for decision making, behavior guidance and emotion regulation, and parents support children with their self-regulatory capacities by providing an environment in which they assist their children in regulating inner feelings and behaviors (Kopp, 1982; Sameroff, 2010). When entering teenage years, adolescents transit into a phase with relative independence as they gradually desire more autonomy from parents: adolescents interact less with parents and more with peers, and are more likely to negotiate with parents about social customs and/or conventions instead of merely following parents' guidance (Steinberg & Silk, 2002). As such, in the teenage years the influence of parenting on the development of self-control may decrease (Farley & Kim-Spoon, 2014; Hay & Meldrum, 2016; Tiberio et al., 2016).

However, while adolescents may feel they are independent and responsible individuals, parents generally remain important figures adolescents turn to for emotional and financial support when needed (Buist, Deković, Meeus, & van Aken, 2002; Li, Delvecchio, Miconi, Salcuni, & Di Riso, 2014). These social changes and developmental transitions in adolescence yield an important question: Is parenting still important for self-control during adolescence? Asking this question for adolescence is all the more important as scientists increasingly recognize that developmental and growth processes that have their beginning in early adolescence continue into the twenties (Sawyer, Azzopardi, Wickremarathne, & Patton, 2018). Additionally, as societal changes lead to delays in adopting adult roles (e.g., life expectancy, longer educational trajectories), research calls for extending our conceptualization of adolescence as ranging from early adolescence to the early twenties (Arroyo, Payne, Brown, & Manning,. 2013; Sawyer et al., 2018). In light of these considerations, researchers have pointed out that the adolescent period covers "a greater proportion of the life course with greater relevance for human development than ever before" (Patton et al., 2018, p. 458). Thus, it would be crucial to take stock of the empirical findings and inform the field about the association between parenting and self-control across the course of adolescence.

The current meta-analysis adds to previous meta-analyses in several important ways. Previous meta-analyses regarding the association between parenting and self-control focused either on young children (Davis et al., 2017; Karreman et al., 2006) or on specific parenting practices or indicators (i.e., parent-child synchrony, Davis et al., 2017; attachment security status, Pallini et al., 2018). Moreover, existing meta-analyses failed to take possible reciprocal effects of self-control on subsequent parenting into account (Sameroff, 2010). Especially during adolescence, it is important to consider the effects of adolescent self-control on parenting, which may play a particularly important role in promoting and maintaining its continuity across adolescence. Indeed, it may well be that early adolescent self-control evokes responses from the parental environment that reinforce the child's tendencies over time (e.g., Caspi & Roberts, 2001). Finally, existing studies did not apply three-level analyses, a novel technique to better distinguish variance at the sample, within-study, and between-study level, to provide a more accurate estimate of the results (Assink & Wibbelink, 2016), such as taking into account dependency between different effect sizes extracted from the same study without losing the rich information of a study that contains multiple effect sizes (Cheung, 2014; Van den Noortgate et al., 2013).

To our knowledge, a comprehensive review synthesizing empirical studies on the association between broad types of parenting and self-control among adolescents is still lacking. This is crucial because the interplay between individual and social environment is at the core of the development of self-control, especially across the adolescent period. Adolescents increasingly become active agents of their own development, and their levels of self-control are likely to evoke responses from their parents generating reciprocal influence of persons and environments (Eisenberg et al., 2005; Sameroff, 2010; Tiberio et al., 2016). In order to clarify whether parenting is associated with self-control across adolescence and identify factors that influence this association, this pre-registered meta-analysis[1] aimed to answer the following questions: (1) Is parenting important to self-control of adolescents aged 10 to 22 years?[2] (2) Does adolescents' self-control influence subsequent parenting? (3) Do theoretical (e.g., types of parenting, age, culture, parent and adolescent gender) and methodological (e.g., report informant, consistency of report informant, and study design) factors included in previous meta-analyses about self-control moderate the magnitude of the association between parenting and self-control in adolescence? Investigating these questions allows us to aggregate diverse individual study results to identify the overall mean effect and examine the role of possible moderators on the magnitude of this effect. Doing so generates insights about self-control development over the course of adolescence and elucidates gaps that should be given attention in future research aiming to understand individual differences in this important capacity.

Conceptualization of self-control

Research on self-control spans the social and behavioral sciences (Duckworth & Steinberg, 2015). The nomenclature of self-control varies by theoretical tradition, with social psychologists and criminologists referring more often to self-control (e.g., Gottfredson & Hirschi, 1990; Tangney, Baumeister & Boone, 2004), while developmental psychologists often refer to concepts such as self-regulation and effortful control (e.g., Bridgett, Burt, Edwards, & Deater-Deckard, 2015; Eisenberg et al., 2003, 2005; Kopp, 1982).

1 we pre-registered this research on https://aspredicted.org/gs2zj.pdf

2 Adolescence is a period connecting early childhood and emerging adulthood. While the adolescent period is considered to begin around 10 years, characterized by the onset of puberty (United Nations Children's Fund, 2011), the end of this developmental period receives less consensus. Recent neuroscientific research has suggested that 21-22 years could be the cut-off age when adolescents become adults (Cohen et al., 2016). Thus, this study focuses on early adolescence to late adolescence ranging from 10 to 22 years.

Although there is considerable dissent in the literature about how to label or define self-control, existing theories and findings generally agree that self-control, self-regulation, and effortful control tap into the same capacity. The common thread going through these concepts is the involvement of voluntary self-governance, an ability one consciously uses to manage one's cognition, emotion, and behavior (Bridgett et al., 2015; Duckworth & Kern, 2011; Nigg, 2017). Evidence from various aspects supports this view. First, studies applying factor structure analyses combining tasks attributed to different conceptualizations of self-control are best presented by a single factor model (Allan, Hume, Allan, Farrington, & Lonigan, 2014; Allan & Lonigan, 2011). Second, a meta-analysis which summarized the convergence of a number of self-control measures derived from different theories, perspectives, and approaches revealed that these measures are moderately convergent (Duckworth & Kern, 2011). Additionally, neuroscientific research showed overlapping neural substrates associated with these concepts (Fan, Flombaum, McCandliss, Thomas, & Posner, 2003; Garavan, Ross, Murphy, Roche, & Stein, 2002). Therefore, in this study, we included self-control as well as these analogous terms, referring to them overall as 'self-control', as done by prior meta-analyses on self-control (Davis et al., 2017; De Ridder et al., 2012; Karreman et al., 2006; Pallini et al., 2018; Vazsonyi, Mikuška, & Kelley, 2017).

The association between parenting and self-control

Parenting is a multifaceted construct containing various terms reflecting different aspects of parenting processes. In this research, we mainly focused on three broad dimensions of parenting (i.e., positive parenting practices, negative parenting practices, and parent-child relationship) in relation to adolescent self-control. Positive parenting refers to parental behaviors that reflect positive control and warmth, such as parental warmth, monitoring, supervision, consistent discipline, parental support and authoritative parenting (Darling & Steinberg, 1993; O'Connor, 2002). Negative parenting refers to behaviors that reflect negative control and hostility, such as harsh parenting, inconsistent discipline, coercive punishment, authoritarian parenting and permissive parenting (Darling & Steinberg, 1993; O'Connor, 2002). Parent-child relationship refers to children's emotional bond with parents (Cassidy, 1994); this construct is also often labeled parent-child attachment (Bowlby, 1969), or parent-child bonding (Gottfredson & Hirschi, 1990) across the literature.

The reasons for us to concentrate on these three broad types of parenting are threefold. First, the parenting literature has primarily focused on two broad categories of parenting, namely parenting behavior/practices (Darling & Stenberg, 1993), which is further divided into positive and negative parenting practices (O'Connor, 2002), and emotional relationship/bond between parent and child (Belsky, 1984; Bowlby, 1969). Second, these three parenting categories correspond to existing theories from various disciplines. For example, the general theory of crime postulates that self-control is nurtured by positive parenting practices (monitoring, consistent discipline) and a close parent-child relationship, while negative parenting practices (excessive punishment, permissive parenting) and weak parent-child relationship undermine self-control development (Gottfredson & Hirschi, 1990). The process model (Belsky, 1984) also suggests that parenting and parent-child relationship are different constructs within the family system that affect child development (Belsky, 1984). Third, as shown in Table 1, we have identified a host of specific parenting terms, with some (e.g., conflict, monitoring, authoritative) much more frequently used to examine the "parenting – self-control" association than others (e.g., neglect, overprotection, alienation). An advantage of focusing on the three broad types of parenting dimensions is that it allows us to group many relevant studies together, ensuring sufficient statistical power. If we focused on each specific term separately, it would not be possible to conduct moderation analyses because many terms would only appear in the literature a few times (e.g., neglect, overprotection, alienation) and statistical power would be low. Hence, in this study we focused on the three types of parenting dimension, a strategy adopted by prior meta-analyses on parenting and child outcomes (e.g., Davis et al., 2017; Karreman et al., 2006; Pallini et al., 2018; Slagt, Dubas, Deković, & van Aken, 2016).

The influence of parenting on self-control

Parents who employ positive parenting strategies provide clear standards for behavior (Sroufe, 1996), monitor and discipline their children's undesirable behavior timely and consistently (Gottfredson & Hirschi, 1990), and guide them to work through problems themselves (Putnam, Spritz, & Stifter, 2002; Strand, 2002), all of which help them gradually internalize others' rules and expectations of what are appropriate behaviors and may foster the development of self-control of adolescents. Many studies linked positive parenting with youth's self-control, generally finding that positive parenting relates to good self-control development in adolescents (e.g., Finkenauer et al., 2005; Hay,

2001; Hope et al., 2003; Özdemir et al., 2013; Vazsonyi & Belliston, 2007). However, it warrants attention that some inconsistent findings emerge in terms of significance and magnitude. For example, in their study Baardstu et al., (2017) found no significant longitudinal associations between positive parenting and self-control over the course of early adolescence.

Parents who use negative parenting strategies are likely to deprive youths of opportunities to figure out self-control strategies independently (Grolnick, McMenamy, & Kurowski, 1999), provide youths with little or no guidance to self-regulate when needed (Baumrind, 1991), monitor and discipline youths' undesirable behavior inconsistently or ineffectively (Gottfredson & Hirschi, 1990), and create a stressful family environment that jeopardizes children's internalization of social rules (Kochanska & Aksan, 1995; Silverman & Ragusa, 1990). Negative parenting therefore provides a context that hampers the development of self-control in adolescence. Consistent with this suggestion, prior research has generally found that negative parenting is related to low self-control (e.g., Brody & Ge, 2001; Cheung & Cheung, 2010; Feldman & Wentzel, 1990; Hallquist, Hipwell, & Stepp, 2015; Simons, Simons, Chen, Brody, & Lin, 2007). Again, however, the evidence is inconsistent. For instance, prior research found that mothers' authoritarian and permissive parenting style and fathers' authoritarian and permissive parenting style were not significantly related to their adolescent children's self-control (Jabagchourian, Sorkhabi, Quach, & Strage, 2014).

For parent-child relationships, parents who successfully establish close bonds foster children to develop better emotional regulation ability, which lays the foundation for further development of self-control (Bowlby, 1969; Cassidy, 1994; Kopp, 1982; Pallini et al., 2018). A close parent-child relationship (in some studies defined as secure parent-child attachment)[3] during adolescence serves as the foundation for parents to monitor, recognize and discipline adolescents' behavior (Gottfredson & Hirschi, 1990) and, in turn, encourages children to share information about their daily activities with their parents (Kerr & Stattin, 2000; Parke, 2004). A close parent-child relationship thereby provides a context that is conducive to parental scaffolding and teaching of self-control. Consistent with this suggestion, numerous studies found that secure attachment is associated with better self-control (e.g., Alvarez-Rivera

3 Attachment during adolescence is usually understood/measured as close parent-child relationship and can be considered as the continuum of childhood attachment rather than the attachment dimensions as by Bowlby & Ainsworth (Armsden & Greenberg, 1987). In this study we therefore used the term "parent-child relationship" instead of "parent-child attachment", but in literature search and coding we categorized relationship and attachment as the same subtype, namely 'parent-child relationship'.

& Fox, 2010; Li et al., 2015; Nie, Li, & Vazsonyi, 2016; Wills, Gibbons, Gerrard, Murry, & Brody, 2003; You & Kim, 2016). Nevertheless, the strength of the association varied considerably. Some studies found that the relation between parent-child relationship and self-control was close to zero (e.g., Jones, Lynam, & Piquero, 2015); some found the relation to be significant but small (e.g., Walters & DeLisi, 2013), and some found the relation to be significant with medium effect size (e.g., Watt & McNulty, 2016).

The influence of self-control on parenting

Adolescents are not passive recipients to parenting behaviors. Over the course of adolescence, their behaviors increasingly influence parenting behaviors. As such, adolescent development can be understood as a transactional or reciprocal process, where environmental factors (e.g., parenting, in this case) affect the development of adolescents (e.g., self-control) while an adolescent's behavior can also evoke certain reactions from his/her environment (for details, see transactional model of development, Sameroff, 2010; social cognitive theory of personality, Bandura, 1999; genotype-environment correlation, Plomin, DeFries, & Loehlin, 1977; ecological systems theory, Bronfenbrenner, 1979).

In parent-child dyads, parenting behaviors (e.g., parental trust and warmth) are related to parents' knowledge about the children (Kerr, Stattin, & Trost, 1999). When parents know their children can exert self-control, resist temptations, and regulate their own behavior, parents are likely to trust their children, grant more autonomy and respond positively to their behavior (Buyukcan-Tetik, Finkenauer, Siersema, Vander Heyden & Krabbendam, 2015). Prior studies involving adolescents and parents have found that adolescents with high levels of self-control elicit trust and warmth from their parents (Bradley & Corwyn, 2007; Buyukcan-Tetik et al., 2015; Tiberio et al., 2016). However, longitudinal research conducted among adolescents also reveals non-significant effects of self-control on parenting over time (Eisenberg et al., 2005; Moilanen, Rasmussen, & Padilla-Walker, 2015).

Similarly, children with good self-control are less impulsive and restless and engage in more socially desirable behavior, which may facilitate parents' relationship with them (Meldrum et al., 2012). Although some studies have supported this relation in adolescents (Bradley & Corwyn, 2007; Otterpohl & Wild, 2015), other studies failed to find such associations in adolescent samples (Meldrum et al., 2012). Conversely, adolescents with poor self-control are likely to engage in delinquent behavior and succumb to temptation, which may spur parents to assert their power by employing harsh or coercive

practices to teach their children a lesson and ostensibly prevent the occurrence of the behavior in the future (Karreman et al., 2006; Kochanska, Aksan, & Koenig, 1995; Silverman & Ragusa, 1990). For instance, prior research found that adolescents with poor self-control are likely to elicit harsh parenting one year later (Brody & Ge, 2001). However, inconsistent findings also emerge. To illustrate, Moilanen et al. (2015), found that adolescents' self-control negatively predicts mothers' but not fathers' authoritarian parenting.

Potential moderators

As shown above, the findings of the association of adolescent self-control and parenting are not consistent. This implies that the association between parenting and adolescent self-control may be moderated by a number of other factors, such as culture, age, and gender. A number of potential moderators are listed below.

Theoretical moderators

Type of parenting. Parenting is commonly categorized as positive and negative in terms of control and warmth (Darling & Steinberg, 1993; O'Connor, 2002), suggesting that positive and negative parenting include both emotional (e.g., affection, warmth) and behavioral (e.g., monitoring and control) components. Close relationship refers to emotional bonding and thus it seems to solely represent the emotional component. Although a close emotional bonding is important to the development of self-control, it is not enough to instill children with self-control without consistent discipline and appropriate monitoring (Gottfredson & Hirschi, 1990). This implies that the three categories of parenting may play a different role in the development of adolescent self-control. Therefore, in this study, we explored whether the three types of parenting relate differently to self-control over the course of adolescence.

Age. As adolescents develop, they become more independent, gain more autonomy from parents, negotiate more about social conventions, and become less attached to their family (Steinberg & Silk, 2002), suggesting that the association of parenting and self-control is likely to decrease over the course of adolescence. Recent research examined the influence of parenting on effortful control from early childhood to early adolescence, revealing that the effects of parenting practices (both positive and negative practices) decreased as children grew older (Tiberio et al., 2016). Moreover, in Vazsonyi and Belliston's (2007) study, the associations between positive parenting (i.e., closeness, support, monitoring) and low self-control were lower among U.S. college students

(r = -.104, -.166, and -.117, respectively) than those among U.S. urban (r = -.212, -.248, and -.219, respectively) and rural (r = -.185, -.326, and -.132, respectively) high school students. These findings, coinciding with the theories on autonomy development of adolescents (e.g., Collins & Steinberg, 2006), led us to hypothesize that the association between parenting and self-control would diminish as adolescents become older.

Culture. Prior research revealed cross-cultural differences in parenting. For instance, Chinese parents are thought to use a more authoritarian style (or harsh parenting) than Western parents (Chao, 1994; Ng, Pomerantz, & Deng, 2014). However, scholars argue that parenting is closely dependent on cultural contexts and therefore any type of parenting, no matter whether it is positive or negative, should be effective in socializing children in a given culture (Fu & Markus, 2014; for on overview, see Smetana, 2017). Some cross-cultural studies directly compared the association between parenting and self-control in adolescents from different cultural and ethnical backgrounds, but yielded mixed evidence. For instance, research by Vazsonyi and colleagues found that the association between positive parenting (e.g., closeness and monitoring) was significantly related to self-control in Swiss, Dutch, Hungarian adolescents, but not in Slovenian or Japanese (for monitoring only) adolescents, and concluded such inconsistencies might be due to cultural differences in parenting (Vazsonyi & Belliston, 2007; Vazsonyi, Trejos-Castillo, & Huang, 2006). In Vazsonyi et al.'s (2016) study performed among Czech adolescents, the researchers found a positive relation between parental monitoring and self-control for Roma but not for non-Roma adolescents. Conversely, Li and colleagues (2015) found that the association between attachment to parents and self-control was largely invariant between Chinese and Italian adolescents. Given these findings, we explored whether culture moderates the "parenting – self-control" relation.

Culture contains multiple dimensions and individualism is one of the most used variables to define cultures (Oyserman, Coon, & Kemmelmeier, 2002). According to Hofstede (2001), some countries are more individualistic than others and he developed an "Individualism Index" to reflect the levels of individualism of a country. In this sense, culture is treated as a continuum instead of a dichotomous category (e.g., Eastern vs. Western; or independent vs. dependent). Moreover, we were aware that a number of studies on self-control and parenting involved several ethnicities within their own country that may be not entirely mapped onto a country's levels of individualism (e.g., Asians residing in U.S.). Thus, we used both Hofstede's individualism score and ethnicity to capture the role of culture. In this study, we explored

whether individualism score of the country where the samples were recruited and adolescent ethnicities would moderate the "parenting – self-control" association.

Adolescent gender. Research on gendered socialization (Gerson, 1985; Hagan, Simpson, & Gillis, 1987; Hayslett-McCall & Bernard, 2002) suggests that the processes shaping self-control may differ by gender. For instance, parents may tolerate certain behaviors (e.g., hang out with friends at night) conducted by boys that would be quickly curtailed if displayed by girls. This suggests that the influence of parenting on self-control could differ between boys and girls. Some studies found that the associations between parenting and self-control were larger for girls than for boys (e.g., Evans, Simons, & Simons, 2012; Larsen et al., 2012; Mandara & Pikes, 2008). However, another line of work suggests that although parents may use different strategies to educate boys and girls, the effectiveness of parental socialization on children's development of self-control is comparable (Beaver, Wright, & DeLisi, 2007; Chapple et al., 2010; Li et al., 2015; Lynskey, Winfree Jr., Esbensen, & Clason, 2000). In this study, we explored whether the association between parenting and self-control differed as a function of proportions of boys and girls in the study sample.

Parent gender. Traditionally mothers are considered to be the main caregiver in the home who provide daily care and are the most important socialization agents (Buist et al., 2002; Munroe, Munroe, Westling, & Rosenberg, 1997; Song, Thompson, & Ferrer, 2009). Yet, some theories suggest that fathers and mothers are equally important to the socialization of children (Gottfredson & Hirschi, 1990). Moreover, it is theorized that despite the traditional role of mothers in the family, fathers also play a significant role in children's adjustment, including the development of control of misbehaviors (Lamb, 2010). However, empirical evidence for these suggestions is mixed. In some studies, the association between parenting and self-control appears stronger for maternal parenting (Intravia, Jones, & Piquero, 2012; Patock-Peckham, Cheong, Balhorn, & Nagoshi, 2001) or for paternal parenting (Feldman & Wentzel, 1990; Morris & Age, 2009), whereas some studies find similar magnitude for both maternal and paternal parenting (e.g., Li et al., 2015; Nie et al., 2016; Özdemir et al., 2013). Given such disparities, we explored whether the "parenting – self-control" association varied as a function of the proportions of mothers and fathers in the study sample.

Methodological moderators

Report informant. Studies use a variety of methods to examine the relationship between parenting and self-control. Part of them utilizes self-report measures, part of them uses other informants (e.g., parent-report, teacher-report), and part of them even employs observational and behavioral methods. Results on the "parenting – self-control" association may vary across informants, because family members as well as teachers and observers may have different experiences or views regarding parent-child interactions and adolescent self-control (Duckworth & Kern, 2011; Lanz, Scabini, Vermulst, & Gerris, 2001). In the present study, we explored whether the relation between parenting and self-control was different among different report informants.

Consistency of report informant. We further examined whether consistency of report informant across constructs may moderate the link between parenting and self-control. Hypothetically, when the two constructs are assessed by the same informant (especially using self-report measures), their correlation is likely to be higher than when the two constructed are assessed by means of different informants (Willems et al., 2018a). Additionally, research found that the correlation between self-reports and other-reports on personality questionnaires is higher than the correlation between self-reports and behavioral tests (Duckworth & Kern, 2011; Harden et al., 2017; Meyer, 2001). In light of this evidence, we explored whether the association between parenting and self-control is stronger when the two constructs were assessed using the same (i.e., consistent) rather than different (i.e., inconsistent) informants.

Study design. Both cross-sectional and longitudinal designs are used to test the association between self-control and parenting, but differences in the magnitude of concurrent versus longitudinal associations are not well quantified. Such comparisons have been done in other meta-analyses focusing on the link between self-control and deviance, with some studies revealing larger effect sizes for cross-sectional than for longitudinal study designs (Pratt & Cullen, 2000) whereas others find no significant differences between designs (Vazsonyi et al., 2017). In this meta-analysis, we tested whether the association between parenting and self-control would be different in magnitude for cross-sectional versus longitudinal studies. Using longitudinal studies, we also explored whether the influence of parenting on later self-control differed from the influence of adolescent self-control on later parenting. This examination, allowed us to pit the effect of parenting on self-control and the effect of self-control on parenting against each other.

METHOD

The PRISMA checklist (Moher et al., 2015) was used as a guideline for the set-up of this meta-analysis. Furthermore, in order to facilitate transparency (Lakens, Hilgard & Staaks, 2016), the aim and hypotheses of the present meta-analysis were pre-registered at AsPredicted ([website is omitted for masked review purpose]), and our full coding sheet including all the obtained effect sizes, moderating variables, and R analysis scripts is available online.

Search of studies

Articles were retrieved through computerized literature search of the electronic databases of the Education Resources Information Center (ERIC), PsychINFO, PubMed, and Web of Science. A literature search was conducted for studies published up to November 2016 with three categories of key phrases used for the search (1) key words containing variables concerning parenting or parent-adolescent relationship (*parent** or *mother** or *father** or *parental* or *maternal** or *attachment** or *family** or *bond**); (2) key words regarding self-control (*self-control* or *self control* or *self-regulation* or *self regulation* or *self-discipline* or *self discipline* or *effortful-control* or *effortful control*[4]*;* and (3) key words focusing on adolescents[5] (*adolescent** or *adolescence* or *teen** or *youth** or *child** or *student** or *undergraduate* or *emerging adult** or *young adult**).

Inclusion criteria

Studies were eligible for this meta-analysis when they met the following criteria. First, the study had to assess the relationship between any type of parenting (e.g., parental warmth, parental harshness) or parent-adolescent relationship (e.g., parental attachment, parental bond) and self-control. Specifically, the study had to report on self-control, or interrelated concepts such as self-regulation, effortful control, or domain-specific forms of control such as impulse regulation. In case no correlations were reported in the article, we contacted the corresponding author.

4 In the pre-registration, we did not include the search term effortful control because we expected our initial terms to yield articles on effortful control. However, during an initial trial of our search, we noticed that important effortful control articles were missing. As a result, we repeated the search including "effortful" and "control" as separate search terms to make our search more inclusive. Also, Prof. M.Bartels and Prof. M. Dekovic were added as co-authors to the paper considering their invaluable insights and collaborations later in the project. Given that we included existing data sets, we did not seek ethical approval in the current work.

5 Key words such as "child" and "youth" were included in the search in order to ensure inclusion of longitudinal studies, focusing on early/middle childhood but possibly also including longitudinal correlations up to adolescence.

Second, the study had to focus on community-based samples, excluding clinical populations with psychological (e.g., cognitive impairments, autism) and/or physical symptoms (e.g., traumatic brain injury, diabetes, asthma), and/or criminal offenders. We are interested in the general population, and clinical groups may influence the magnitude or direction of effect sizes (Rothbaum & Weisz, 1994).

Third, the mean age of the participants in the study had to fall within the age range of adolescent period used in the current study (i.e., 10 – 22 years). Twelve to eighteen is commonly seen as the age range of adolescence. However, we decided to broaden this range for two reasons. First, the beginning age of adolescence is related to puberty and WHO (2017) has considered that age 10 can be seen as the starting age of adolescence because of the earlier onset of puberty than before. Second, we consider age 22 as the upper bound of adolescence because in this new era youths have more time to develop instead of rushing for reproduction or making a living (National Research Council, 2015). Additionally, a recent study suggests that age 21 to 22 can be seen as the cut-off age when adolescents become adults in terms of brain maturity (Cohen et al., 2016). In the case of longitudinal designs, the study had to assess at least one of the constructs (i.e., parenting or self-control) during adolescence. For example, in a longitudinal study that assessed parental warmth and self-control at age 8, age 12, and age 14, the concurrent and longitudinal correlations within and between age 12 and age 14 were included. Longitudinal correlations between age 8 and age 12 and between age 8 and age 14 were also included, but concurrent correlations at age 8 were excluded.

Fourth, the study had to be published in English in a peer-reviewed journal with full-text downloadable. We did not include unpublished work, review articles, book chapters, dissertations and conference abstracts as findings in these are often subsequently published in peer-reviewed journals. Inclusion of only peer-reviewed articles has been widely accepted in prior meta-analyses (e.g., Karreman et al., 2006; Lovejoy, Graczyk, O'Hare, & Neuman, 2000; Slagt et al., 2016). Moreover, research has shown that meta-analyses that include unpublished studies are just as likely to find evidence for publication bias as those that do not (Ferguson & Brannick, 2012).

Selection procedure

The initial search in the databases yielded 6,792 hits, after removing duplicates. The first two authors screened all abstracts independently, selecting articles for full text reading. This resulted in 814 potentially relevant articles. These

articles were carefully screened to determine whether they met the inclusion criteria. A number of articles were excluded because: they did not include an appropriate measure of parenting and/or self-control (k = 252); the study consisted of a clinical population or a population in the wrong age range (k = 94); no full-text in English was available (k = 76); the article was not published as journal articles (k = 69); and no correlation table was available (k = 156). For the latter 156 articles, the corresponding authors were contacted by e-mail to request additional information. Some authors declined our invitation because they did not have access to the data anymore (6%), some authors could not be contacted because no valid e-mail address was found (12%), and others provided us with the necessary correlations (15%, yielding k = 24 additional articles to include). Most of our e-mails remained unanswered (67%). Finally, 191 studies met the selection criteria and were included in the meta-analysis. See Figure 1 for the PRISMA flowchart depicting the full search and inclusion process.

Coding of the studies

We developed a detailed coding scheme based on guidelines proposed by Lipsey and Wilson (2001), recording study descriptors and study characteristics potentially moderating the relation between parenting and self-control in adolescence. Study descriptors included basic information for all studies, such as author names, year of publication, article title, details on data collection, and sample size. Study characteristics possibly moderating the relation between parenting and self-control in adolescence were grouped into two moderator categories: moderators of theoretical interest and methodological characteristics.

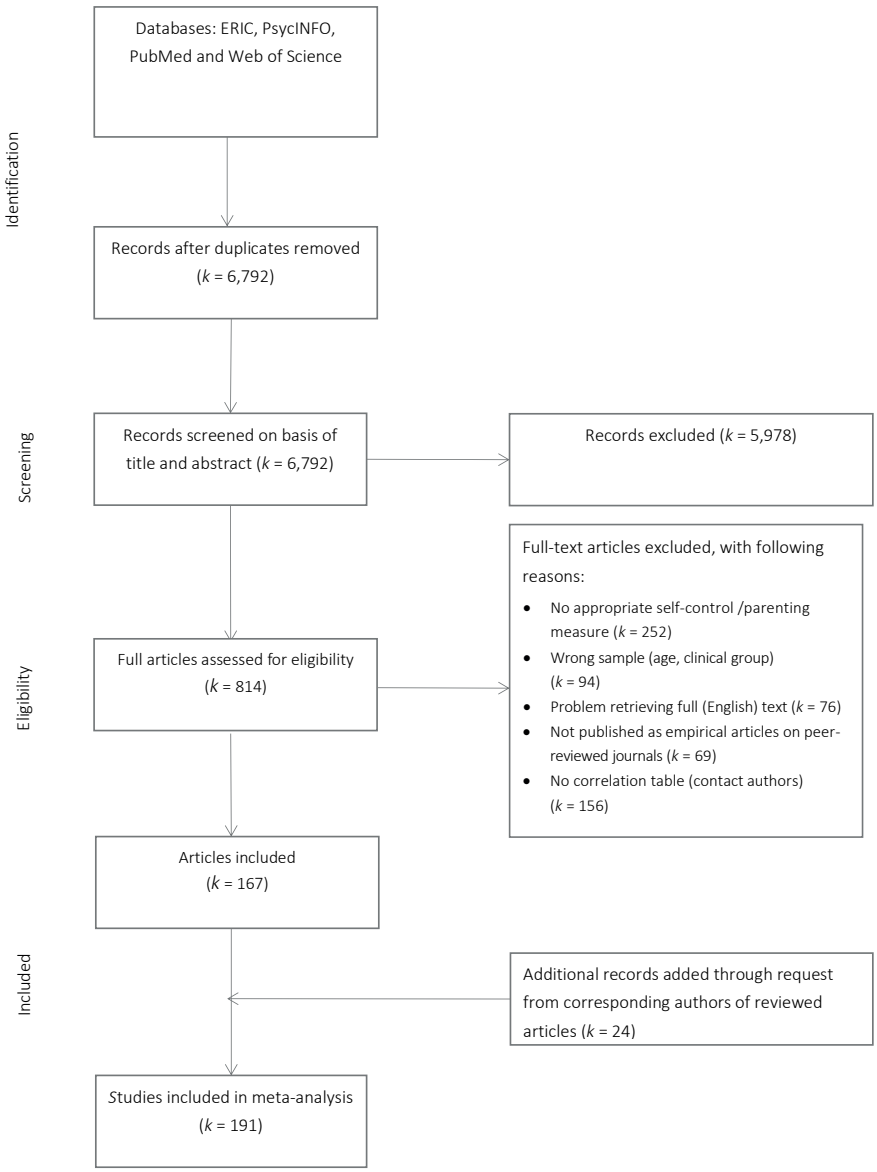

Figure 1. PRISMA flowchart used to identify studies for detailed analysis of parenting and self-control.

Theoretical moderators

Type of parenting*.* To assess possible moderating effect of parenting types, parenting practices and parenting characteristics were subdivided into the three categories (Davis et al., 2017; Doyle & Mariewicz, 2005; Gallarin & Alonso-Arbiol, 2012; Karavasilis et al., 2003; Hoeve et al., 2009; Karreman et al., 2006; Slagt et al., 2016): 1) positive parenting, including supervision, support, autonomy sensitivity, involvement, monitoring, authoritative parenting, warmth, positive expression etc.; 2) negative parenting, including harsh parenting, neglect, rejection, negative expressions, authoritarian, permissive, etc.; and 3) parent-child relationships, including social bonds, closeness, attachment, security, etc.. Studies were coded accordingly (coded categorically 1 = *positive parenting,* 2 = *negative parenting*, 3 = *relationship*). See Table 1 for a detailed overview of the subdivision of parenting types.

Age. We coded age continuously. Some studies did not report age, but school grade. If this was the case, we took the average age of that grade. For example, in grade six in the USA children are on average between age 11-12, and we therefore considered 11.5 years as a mean age for this sample.

Culture*.* As mentioned above, we used individualism index and ethnicity to represent culture. We coded the level of individualism of the country where the data were collected according to Hofstede's individualism score (see www.hofstede-insights.com). The score is a continuous index with higher scores for more individualistic societies (e.g., an index of 91 for the USA) and lower scores for more collectivistic societies (e.g., an index of 17 for Taiwan).Regarding ethnicity, we coded ethnicity of adolescents (coded categorically, 1 = *balanced* (i.e., no ethnicity exceeded 60% of the sample)*,* 2 = *the sample consisted of more than 60% Caucasian,* 3 = *the sample consisted of more than 60% African or African-American,* 4 = *the sample consisted of more than 60% Asian or Asian American*, 5 = *the sample consisted of more than 60% Hispanic,* 6 = *other).*

Adolescent gender. We coded adolescent gender according to the proportion of boys and girls included in the sample (coded categorically, 1 = *overall balanced* (the percentage of boys or girls of the sample ranging between 40% and 60%)*,* 2 = *>60% boys*, 3 = *>60% girls*).

Parent gender. Studies were coded as to whether the parenting referred specifically to adolescents' mothers or to adolescents' fathers. Often, however, parenting measures assessed 'parenting' in general, and did not specifically mention whether the measure focused on mothers, fathers, or both parents. As a result, we categorized the variable as follows: 1 = *greater proportion of*

mothers (> 60% of the sample), 2 = *greater proportion of fathers* (> 60% of the sample), 3 = *both parents, no clear proportion*.

Multilevel analysis allows researchers to include multiple effect sizes from one study while simultaneously taking dependency into account. As such, categories of positive and negative parenting, and parent-child relationship were not mutually exclusive, with studies contributing effects sizes to multiple categories. Similarly, studies contributed to multiple ages if the design of the study was longitudinal, and parent gender if effects of parenting on adolescent self-control were given separately for mothers and fathers. Nevertheless, the multilevel analyses allowed us to diminish the bias caused by the studies that provide multiple inputs.

Methodological characteristics

Report informant. Studies were coded for the informant of the parenting measure (coded categorically, 1 = *adolescent self-report,* 2 = *other-report, such as parent, mother, father, etc.*, 3 = *observation*, *4 = composite, combining measures of multiple informants or assessment modalities),* for the informant of the self-control measure (coded categorically, 1 = *adolescent self-report,* 2 = *other-report, such as parent, mother, father, etc.*, 3 = *observation*, *4 = composite, combining measures of multiple informants or assessment modalities)*.

Consistency report informant. Based on more detailed information of the informants, we coded whether the parenting and self-control measures were assessed by the same informant to assess report informant consistency (coded categorically, 1 = *consistent,* 2 = *inconsistent*). Attention was paid to the elements that comprised of the composite score. For example, if parenting and self-control were both composite scores combined from self-report and mother-report, then we considered the informant as consistent. If one composite score was combined from self-report and mother-report whereas the other combined self-report and teacher-report, we considered the informant as inconsistent.

Study design. Study design was coded as a categorical variable, including whether the effect size between parenting and self-control was derived from cross-sectional or longitudinal studies (1 = *cross-sectional,* 2 = *longitudinal*). Additionally, for longitudinal studies we included effect sizes where parenting was measured first, and self-control was measured some time later (Parenting (P) → Self-control (SC)). Similarly, we included effect sizes where self-control was measured first, and parenting was measured some time later (Self-control (SC) → Parenting (P)). As a result, we coded whether the effect size referred to the influence of parenting on self-control or the influence of self-control on

parenting (coded categorically, 1 = *from parenting to self-control, P → SC*, 2 = *from self-control to parenting, SC → P*).

Inter-rater agreement

Of the 814 eligible articles, 20% were randomly selected to be double coded by the first two authors. Intra-class correlation (for continuous variables) and Cohen's Kappa (for categorical variables) were calculated. Intra-class correlations for continuous variables were high, ranging from 0.78 (for age) to 1.00 (for individualism score). Cohen's Kappa for the categorical ranged from 0.91 (for including/excluding studies) to 1.00 (for study design, effect size direction, and informant parenting). To resolve disagreement, in-depth reading and discussion followed based on the content of the article. Together, these results showed good inter-rater reliability reflecting a good agreement for the study characteristics between the two independent raters.

Effect sizes

In order to investigate the magnitude of the relationship between parenting and self-control, Pearson's correlation coefficients *r*s were obtained for all included studies (Lipsey & Wilson, 2001). Zero-order correlation coefficients are bivariate estimates typically obtained from each empirical study's correlation matrix, or requested from authors if none was provided in the full-text. To ensure similar direction of effects, effect sizes were recoded if 1) parenting pertained to negative dimensions, and 2) self-control was measured using a scale of 'lack of self-control', or 'low self-control'. We used Fisher's *r* to *z* transformation, converting the effect size estimation from each association into an ES_Z score to correct for skewness in the sampling distribution of *r* (Lipsey & Wilson, 2001). This ES_Z scores is assumed to approach normality, which is necessary for the accurate determination of mean effect size estimates and for unbiased tests of statistical significance. As a result, ES_Z were included in the analyses, and transformed back to Pearson's *r* for interpretation purposes (Field, 2001; Lipsey & Wilson, 2001)[6]. For moderator analyses, categorical variable categories were transformed to *k*-1 dummy variables through binary coding (Assink & Wibbelink, 2016; Lipsey & Wilson, 2001).

6 The Fisher's transformation of *r* was done using the following formula: $ES_{Zr} = \frac{1}{2}\log_e\left[\frac{1+r}{1-r}\right]$. Any ES_{Zr} can be transformed back into standard correlation form using the inverse of the ESZr transformation using the following formula: $r = \frac{e^{2ESZr}-1}{e^{2ESZr}+1}$ (see Field, 2001; Lipsey & Wilson, 2001).

Publication bias

Previous research consistently showed that non-significant studies are more likely to be rejected for publication, or remain unsubmitted by authors (Torgerson, 2006; Lipsey & Wilson, 2001). This publication bias may result in inflated effect sizes and restricted range of values in meta-analyses (Rosenthal, 1979). Therefore, it is important to statistically assess the possible influence of publication bias, before interpreting the final results. In the present study, we handled this problem by applying a funnel plot, plotting the distribution of each individual study's effect size on the horizontal axis against its precision expressed in standard errors on the vertical axis (Torgerson, 2006). If a publication bias affects the data, an asymmetrical funnel plot is to be expected (Begg, 1994). Additionally, Egger's test was applied to test the significance of the asymmetry of the plot, providing more precise information on the possible presence of publication bias (Egger, Smith, Schneider, & Minder, 1997). When this test yields significant results, sensitivity analyses were conducted by applying the trim and fill method, correcting for the asymmetric plots by imputing missing effect sizes through a number of iterations (Duval & Tweedie, 2000a, 2000b). Imputing non-existing effect sizes is, however, controversial and effect sizes produced by the trim and fill analyses should be interpreted with caution (Sutton, Duval, Tweedie, Abrams, & Jones, 2000).

Data analyses

All analyses were conducted in the Metafor package in the open-source statistical R environment version 3.4.2 (Viechtbauer, 2010; R Core Team, 2017). Most studies reported on multiple effect sizes. For example, some studies included longitudinal data (yielding effect sizes for different time points), different raters (resulting in effect sizes separately for mother and father report), and effect sizes separately for boys and girls. It is likely that these effect sizes from the same study are more similar than effect sizes from different studies, because they rely on the same sample, data collection and sampling methods. When using nested effect sizes, however, the assumption of traditional meta-analyses that observations are independent and error terms are uncorrelated is violated (Lipsy & Wilson, 2001; Rosenthal, 1984). Not taking into account this dependency can result in a biased result, as it may create artificially narrow confidence intervals and shrunken standard errors favoring statistical significance (Hox, Moerbeek & Van de Schoot, 2010).

Strategies applied to handle the aforesaid dependency problem include selecting one effect size from each study, averaging effect sizes within studies

or simply ignoring the dependency of effect sizes (Lipsey & Wilson, 2001). However, more recently multilevel meta-analysis has been suggested as a more preferable tool, as it takes into account dependency while including all available effect sizes resulting in maximum information and greater statistical power (Assink & Wibbelink, 2016; Hendriks et al., 2017; Hox et al., 2010; Van den Noortgate, López-Lopez, Marín-Martínez, & Sánchez-Meca, 2013). Three level models apply when groups are nested within clusters, and thus are not independent from one another. In our case, we have variance at the effect size level (level 1) that are nested within a sample (level 2, e.g. effect sizes based on Add health data), with also variance between studies (level 3, taking into account effect sizes to vary between studies). Incorrectly modelling this dependency in the data will likely result in biased standard errors, making incorrect inferences about the relationships being studied (Viechtbauer, 2013). Accordingly, we applied a three-level model to account for the three sources of variance: (1) level 1 takes into account sampling variance of the effect sizes, (2) level 2 takes into account variance between effect sizes from the same sample, allowing effect sizes to vary *within studies,* (3) level 3 takes into account variance between studies, allowing effect sizes to vary *between studies* (Hox et al., 2010; Van den Noortgate et al., 2013). Additionally, since parameter estimates from different levels of analyses are not independent in this multilevel approach, no greater weight will be placed on studies with more effect sizes. Thus, a study that, for example, includes 10 effect sizes will not contribute 10 times more to the mean effect size compared to those only has one effect size (Van den Noortgate et al., 2013). Overall, multilevel modeling allows including effect sizes based on the same sample, providing more precision in estimating mean effect sizes while simultaneously modeling the nestedness of the data (Cheung, 2014; Van den Noortgate et al., 2013).

The current three-level analysis was conducted in three stages. First, the overall mean effect sizes were estimated to assess the strength of the association between parenting and self-control in adolescence. Second, we applied a likelihood ratio test to assess between-study and within-study heterogeneity. Important to note is that the level of 'study' entails dataset. For example, multiple papers are based on the Add Health dataset (http://www.cpc.unc.edu/projects/addhealth). Accordingly, for the multilevel analyses we gave studies using the same dataset (e.g. all published studies using Add Health data) the same 'study ID', clustering them as if they were all from one published study. This allowed us to take into account this dependency, referring to the included studies as number of independent studies.

Third, if there was evidence for heterogeneity in effect sizes (presented as a Q_E which, when significant, indicates heterogeneity among effect sizes), moderation analyses were conducted for moderators of theoretical interest and methodological characteristics. In order to obtain reliable results, moderator analyses were only conducted if each category contained at least five studies (parameter estimates are poor when number of studies is very small, Weiss et al., 2017).

The three-level analyses were conducted according to the three-level random effects model guidelines formulated by Wibbelink and Assink (2016), using the restricted maximum likelihood procedure for parameter estimation, and performed with the Metafor package in the open-source statistical software R version 3.4.2 R environment (Viechtbauer, 2010; R Core Team, 2017). Moreover, we used G*Power (version 3.1.9.2, Faul, Erdfelder, Lang, & Buchner, 2007) to calculate the sample size needed for future research to obtain the mean correlation found in this research. Sample sizes for obtaining four levels of power (i.e., .80, .90, .95, & .99) with the alpha level of .05 were recommended.

RESULTS

Descriptive statistics

The present meta-analysis included 191 articles, reporting on 159 independent studies and comprising of 1,540 effect sizes. All studies included in the meta-analysis are marked with an asterisk in the reference section. The overall sample size is *N*=164,459, with sample sizes of studies ranging between *N* = 47 (Samuelson, Krueger, & Wilson, 2012) and *N* = 19,810 (Barnes & Morris, 2012), and a mean age of 13.92 years. The overall sample size was calculated by summing the largest sample size within unique samples when more than one effect size was collected. Publication year of the included studies ranged between 1990 and 2016, with the number of studies published annually represented in Figure 2. Most studies included both boys and girls (80%), with some studies focusing specifically on boys (7%), some specifically focusing on girls (9%), and some studies not providing gender descriptive (4%). Some studies focused on parenting in general, without differentiation between mothers and fathers (44%), some specifically focused on maternal parenting (40%), and few studies specifically focused on paternal parenting (16%). Of all included effect sizes, 53% focused on positive parenting, 33% on negative parenting, and 14% on parent-child relationships (see supplemental table 1 for details on parenting dimensions).

Table 1. Details on parenting dimensions, numbers representing number of effect sizes within each category

Parenting Term	Positive	Negative	Relationship	Total
Authoritative	139			
Monitoring	137			
Support	133			
Responsive discipline	95			
Warmth	81			
Supervision	53			
Positive control	46			
Involvement	33			
Positive expressivity	25			
Sensitivity	24			
Acceptance	21			
Autonomy	17			
Cohesion	6			
Conflict		117		
Authoritarian		97		
Psychological negative control		81		
Harshness		78		
Permissive		44		
Abuse		26		
Rejection		23		
Hostility		16		
Withdrawal		14		
Negativity		10		
Coercion		7		
Attachment			108	
Relationship			47	
Bond			21	
Closeness			16	
Communication			12	
Trust			8	
Alienation			5	
Total number of effect sizes				*1540*

Note: The associations between these dimensions and self-control are presented in Supplemental Table 1.

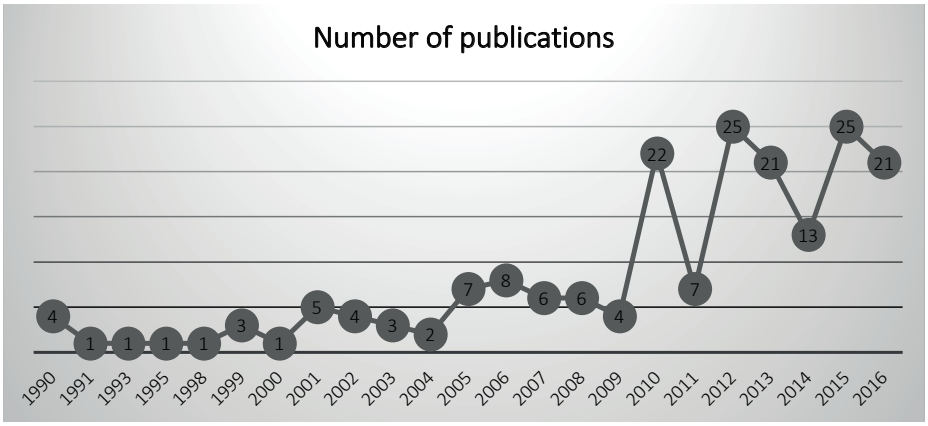

Figure 2. Number of included studies published annually.

Table 1 presents the details on parenting categories. It shows that positive parenting is predominantly comprised of authoritative parenting, monitoring, and support, whereas negative parenting is comprised of conflict, authoritarian parenting, psychological negative control, and harsh parenting, and, to a lesser degree, of parental coercion and withdrawal. Finally, parent-child relationship is mostly comprised of attachment while alienation and trust from the attachment scale make up the smallest percentage of this category.

Studies were conducted worldwide, including studies from Australia, Canada, China, India, Mexico, with most effect sizes retrieved from the USA (62%), South-Korea (10%), Switzerland (6%), and The Netherlands (4%) (see Figure 3 for a graphical representation of the countries represented in the present meta-analysis). Hofstede's individualism indices ranged between 17 (Taiwan) / 18 (South-Korea), and 90 (Australia) / 91 (USA), with k = 28 independent studies reporting on countries with an individualism score below 50 and k = 129 studies reporting on countries with an individualism score above 50.

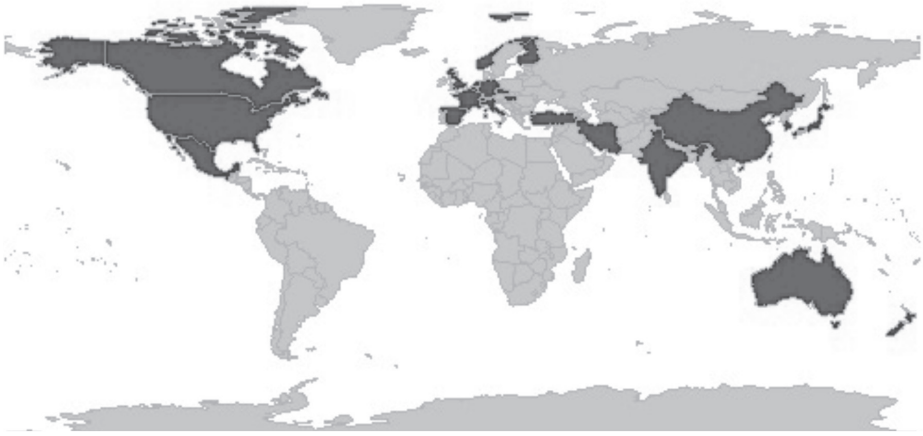

Figure 3. Graphical representation of countries represented in the meta-analysis

The present meta-analysis included both cross-sectional (57%) and longitudinal (43%) effect sizes. Of the longitudinal effects, 56% measured parenting first and self-control some time later (P → SC), and 44% measured self-control first and parenting some time later (SC→ P). Longitudinal studies ranged from 1 month to 13 years, with most studies reporting on a 0-1 year time lag (57%) or a 1-2 year time lag (23%), and others reporting on a 2-3 year time lag (5%), 3-4 year time lag (13%), 4-5 year time lag (2%), and more than 5 year time lag (16%). Thus, the present meta-analysis included: 1) cross-sectional effect sizes 2) longitudinal P → SC effect sizes, where parenting predicted subsequent self-control and, 3) longitudinal SC → P, where adolescent self-control predicted subsequent parenting. These three groups describe different patterns of effects and should be treated individually. Therefore, we assessed overall effects of these three groups separately followed by statistical analyses testing whether these effects statistically differed. Of the 1,540 effect sizes, 876 concerned cross-sectional associations, 373 concerned longitudinal P → SC associations, and 291 concerned longitudinal SC → P associations, respectively.

Overall effects

Cross-sectional association*.* The overall effect size of cross-sectional studies was statistically significant (ES_Z = 0.207, S.E. = 0.010, t = 20.165, p < .001, 95% CI = [0.187, 0.227]), with substantial heterogeneity (Q_E(875) = 13140. 584, p < .001). Using inverse version of the Fisher's (1921) r-to-z formula, we transformed this effect size back to *Pearson r* for interpretation purposes. We

found that the cross-sectional association between parenting and self-control in adolescence was r = .204 (95% CI = [.185, .223]).

Longitudinal associations: parenting → self-control. The overall effect size of longitudinal P → SC was also statistically significant (ES_Z = 0.158, S.E. = 0.015, t = 10.238, p < .001, 95% CI = [0.128, 0.188]), with substantial heterogeneity (Q_E(372) = 3349.047, p < .001). We found that the longitudinal association where parenting was measured first and self-control later was r = .157 (95% CI = [.127, .186]).

Longitudinal associations: self-control → parenting. For longitudinal SC → P, the overall effect size was also statistically significant (ES_Z = 0.156, S.E. 0.022, t = 7.123, p <.001, 95% CI= [0.113, 0.199], with substantial heterogeneity (Q_E(290) = 2293.718, p < .001). The longitudinal association where self-control was measured first and parenting later was r = .155 (95% CI = [.113, .196]).

Comparison between cross-sectional and longitudinal effect sizes. As reported above, there were some observed differences in the effect sizes between cross-sectional and longitudinal studies, and therefore we tested whether this difference was statistically significant. Applying three-level analyses, we found an overall significant difference between the three groups F(2, 1,537) = 26.14, p <.001, and significant heterogeneity in effect sizes Q_E(1537) = 1,878.35, p < .001. As shown in Table 2, the associations for longitudinal P → SC were significantly smaller than cross-sectional associations. Similarly, associations for longitudinal SC → P were also significantly smaller than cross-sectional associations. Results showed no significant difference in the associations between longitudinal P → SC and SC → P.

Subsequently, analyses for these three groups were conducted separately. First, a random-effect model was utilized to calculate variance at the sampling, within-study, and between-study levels. Second, multiple potential moderators were tested individually. Third, a multiple-moderator model including all the significant moderators was performed to control for the associations among moderators. Finally, we also checked publication bias using the Funnel plot (Egger et al., 1997).

Table 2. Comparing cross-sectional and longitudinal effect sizes

	ES_z	S.E.	t	95% CI	*p*
Δslope cross-sectional (vs. P → SC)	-0.050	0.008	-6.228	[-0.066 , -0.034]	< .001
Δslope cross-sectional (vs. SC → P)	-0.052	0.009	-6.058	[-0.068 , -0.035]	< .001
Δslope P → SC (vs. SC → P)	-0.002	0.009	-0.174	[-0.019, 0.016]	.862
Q_E (df)	18783.35 (1537), p < .001				
Omnibus test	F(2,1537) = 26.136, p < .001				
Variance within study	.008, p < .001				
Variance between studies	.010, p < .001				
# ES	1540				

Cross-sectional studies

Variance of the overall effect size. The variance at the within-study level and the between-study level were both significant (p<.001). Follow-up analyses found that variance at the sampling, within-study, and between-study level was 5.13%, 45.11%, and 49.76%, respectively. Hunter and Schmidt (1990) proposed that heterogeneity can be considered as substantial, if less than 75% of the variance can be attributed to the sampling variance and that in this case examination of the moderating effects of the study and/or effect size characteristics on the overall effect can be fruitful. In our study, only 5.13% variance was explained by the samples, indicating that continuous exploration of potential moderators is meaningful.

Moderator analyses. Considering the large statistical power **(***N*=164,459), we were confident to assess potential moderators of theoretical and methodological interest (see Table 3). Regarding the moderators of theoretical interests, the only significant moderator was type of parenting (Q_E(873) = 12763.277, p < .001; Omnibus test: F(2, 873) = 3.483, p = .031). None of the other moderators of theoretical interest, including cultural characteristics (ethnicity, Hofstede's individualism), parent gender (i.e., whether the effect refers to mother or father), adolescent gender (i.e., whether the effect refers to boys and girls), or age of adolescents[7] was significant.

7 We were also interested to see whether age could show a non-linear pattern. To this end, we checked whether the squared and/or cubical age served as a significant moderator. However, our results showed no significant quadratic (Q_E (830) = 10384.754, p < .001; Omnibus F(1, 830) = 1.698, p = .193) or the cubic pattern (Q_E (830) = 10361.892 p < .001; Omnibus F(1, 830) = 1.693, p = .194).

Table 3. Cross-sectional associations: the Q_E statistics testing residual heterogeneity, and the Omnibus to test the effect of the moderators

Moderator	Q_E (df)	*p*	*Omnibus test*	*p*
Theoretical moderators				
Age	10420.319 (830)	<.001	$F(1, 830) = 1.632$	.202
Ethnicity of adolescents	9503.564 (722)	<.001	$F(5, 722) = 1.286$	.268
Hofstede's individualism	12883.360 (862)	<.001	$F(1, 862) = 0.300$	.584
Type of parenting	12763.277 (873)	<.001	$F(2, 873) = 3.483$	.031
Parent gender	13035.444 (865)	<.001	$F(2, 865) = 2.743$	.065
Adolescent gender	10777.295 (840)	<.001	$F(2, 840) = 1.443$	.237
Methodological moderators				
Informant parenting	13034.230 (871)	<.001	$F(3, 871) = 5.172$	.002
Informant self-control	12859.772 (872)	<.001	$F(3, 872) = 5.068$	.002
Consistency informants	13104.212 (874)	<.001	$F(1, 874) = 15.043$	< .001

Pertaining to the moderators of methodological interest, all the three moderators in this category were significant: report informant of parenting measure ($Q_E(871) = 13034.230$, $p < .001$; Omnibus test: $F(3, 871) = 5.172$, $p = .002$), report informant of self-control measure ($Q_E(872) = 12859.772$, $p < .001$; Omnibus test: $F(3, 872) = 5.068$, $p = .002$), and consistency of the report informant of the parenting and self-control measures ($Q_E(873) = 13104.212$, $p < .001$; Omnibus test: $F(1, 874) = 15.043$, $p < .001$).

Significant moderators. Based on the significant moderators found above, follow-up comparison was conducted and the results are summarized in Table 4. Regarding the type of parenting, we found that the effect sizes for the "positive parenting – self-control", "negative parenting – self-control" and "parent-child relationship – self-control" associations were all significant. Results of further comparison suggested that the relationship for negative parenting was significantly smaller than that for positive parenting.

Regarding the informant of parenting measure, we found that the effect sizes of the relationship between parenting and self-control were all significant when parenting was measured using adolescent self-report, other-report, observation, and composite measures. Results of further comparison indicated that effect sizes for studies using composite measures were significantly larger than those using self-report, other-report, and observation, and that

effect sizes for studies using observation measure to assess parenting were significantly lower than those using adolescent self-report and other-report.

With respect to the informant of self-control measures, we found that effect sizes of the relationship between parenting and self-control were all significant when self-control was assessed using adolescent self-report, other-report, observation, and composite measures. Follow-up comparison indicated that effect sizes of studies using composite measures to assess self-control were significantly larger than those using adolescent self-report, other-report, and observation, and that effect sizes of studies using observation measures to assess self-control were significantly lower than those using adolescent self-report.

For the consistency of the report informant on parenting and self-control measures, effect sizes were both significant for studies using consistent and inconsistent report informant. Results of follow-up comparison showed that effect sizes of studies using inconsistent report informants were significantly lower than those using consistent report informants.

Multiple moderator model. According to Hox et al. (2010), moderators may be interrelated, possibly causing multicollinearity problems in the analyses. To overcome this, a multiple moderator model which included all significant moderators found in the individual moderation test above was performed. The results of this model are summarized in Table 5. Omnibus test showed a significant results ($F(9, 865) = 6.157$, $p < .001$), suggesting that at least one of the regression coefficients of the moderators significantly deviated from zero. These results indicated that negative parenting (vs. positive parenting), composite measure of self-control (vs. adolescent self-report), and inconsistent report informant (vs. consistent report informant) had unique moderating effects on the relationship between parenting and self-control.

Publication bias*.* Considering our large sample size, and that, for numerous studies, the association between parenting and self-control was not the primary research interest, we assumed little influence of publication bias. To statistically check this assumption, we inspected funnel plot using Fisher's z transformations (see Figure 4a.), and applied Egger's regression test (Egger et al., 1997; Torgerson, 2006). Results of regression test for Funnel plot asymmetry found that there was no significant asymmetry, $z = -1.506$, $p = .132$, suggesting that no significant publication bias was detected for the results found above.

Table 4. Cross-sectional associations: significant moderators

Moderators	***#ES***	***ES_z***	***SE***	***t***	***95% CI (ES_z)***	***p***	***r***
Type of parenting	876						
Positive parenting	446	0.213	0.011	18.826	[0.191, 0.235]	< .001	.210
Negative parenting[a]	290	0.188	0.013	14.864	[0.163, 0.212]	< .001	.186
Parent-child relationship	140	0.220	0.016	13.676	[0.188, 0.252]	< .001	.217
ΔSlope of positive (vs. negative)		-0.025	0.010	-2.444	[-0.045, -0.005]	.015	
ΔSlope of positive (vs. parent-child relationship)		0.007	0.016	0.458	[-0.024, 0.038]	.647	
ΔSlope of negative (vs. parent-child relationship)		0.032	0.017	1.907	[-0.001, 0.066]	.057	
Report informant of parenting measure	875						
Adolescent self-report	622	0.206	0.011	19.041	[0.185, 0.228]	< .001	.203
Other-report	123	0.199	0.016	12.264	[0.167, 0.231]	< .001	.196
Observation	46	0.114	0.032	3.574	[0.051, 0.176]	< .001	.114
Composite	84	0.276	0.030	9.060	[0.216, 0.336]	< .001	.269
ΔSlope of adolescent self-report (vs. other-report)		-0.007	0.015	-0.490	[-0.036, 0.021]	.625	
ΔSlope of adolescent self-report (vs. observation)		-0.093	0.032	-2.867	[-0.156, -0.029]	.004	
ΔSlope of adolescent self-report (vs. composite)		0.070	0.032	2.185	[0.007, 0.132]	.029	
ΔSlope of other-report (vs. observation)		-0.086	0.034	-2.536	[-0.152, -0.019]	.011	
ΔSlope of other-report (vs. composite)		0.077	0.034	2.259	[0.010, 0.143]	.024	
ΔSlope of observation (vs. composite)		0.162	0.042	3.906	[0.081, 0.244]	< .001	

Report informant of self-control measure	876						
Adolescent self-report	546	0.203	0.011	18.667	[0.182, 0.224]	< .001	.200
Other-report	251	0.188	0.017	11.161	[0.155, 0.221]	< .001	.186
Observation	23	0.121	0.039	3.092	[0.044, 0.198]	.002	.120
Composite	56	0.297	0.029	10.291	[0.240, 0.353]	< .001	.289
ΔSlope of adolescent self-report (vs. other-report)		-0.015	0.017	-0.918	[-0.048, 0.017]	.359	
ΔSlope of adolescent self-report (vs. observation)		-0.082	0.039	-2.08	[-0.159, -0.005]	.038	
ΔSlope of adolescent self-report (vs. composite)		0.094	0.030	3.145	[0.035, 0.152]	.002	
ΔSlope of other-report (vs. observation)		-0.067	0.038	-1.736	[-0.142, 0.009]	.083	
ΔSlope of other-report (vs. composite)		0.109	0.033	3.313	[0.044, 0.173]	< .001	
ΔSlope of observation (vs. composite)		0.176	0.048	3.635	[0.081, 0.270]	< .001	
Consistency of report informant	875						
Consistent	537	0.223	0.011	19.785	[0.201, 0.245]	< .001	.219
Consistent	338	0.168	0.015	11.475	[0.139, 0.196]	< .001	.166
ΔSlope of consistent report informant (vs. inconsistent)		-0.056	0.014	-3.903	[-0.084, -0.028]	< .001	

Note: To ensure similar direction of effects, effect sizes were recoded if 1) parenting pertained to negative dimensions, and 2) self-control was measured using a scale of 'lack of self-control', or 'low self-control'. As such, for the negative parenting dimensions correlations appear positive, but reflect a negative correlation between negative parenting and self-control.

Table 5. Cross-sectional association: results for the multiple moderator model

Moderator variables	**β (SE)**	**95% CI**	***t***	***p***
Intercept	.221 (.013)	[.196, .246]	17.389	< .001
Type of parenting: negative parenting (vs. positive parenting)	-.028 (.010)	[-.048, -.007]	-2.675	.008
Type of parenting: parent-child relationship (vs. positive parenting)	.005 (.016)	[-.026, .036]	0.305	.760
Report informant of parenting: other-report (vs. self-report)	-.008 (.015)	[-.037, .021]	-0.557	.578
Report informant of parenting: observation (vs. self-report)	-.058 (.034)	[-.125, .009]	-1.713	.087
Report informant of parenting: composite (vs. self-report)	.050 (.034)	[-.017, .117]	1.465	.143
Report informant of self-control: other-report (vs. self-report)	.013 (.017)	[-.021, .047]	0.754	.451
Report informant of self-control: observation (vs. self-report)	-.079 (.041)	[-.158, .001]	-1.936	.053
Report informant of self-control: composite (vs. self-report)	.126 (.033)	[.061, .191]	3.829	< .001
Consistency of report informant: inconsistent (vs. consistent)	-.069 (.016)	[-.099, -.038]	-4.422	< .001
Omnibus test:	$F(9, 865) = 6.157$, $p < .001$			
Variance within study	.090, $p < .001$			
Variance between studies	.110, $p < .001$			
# ES	875			

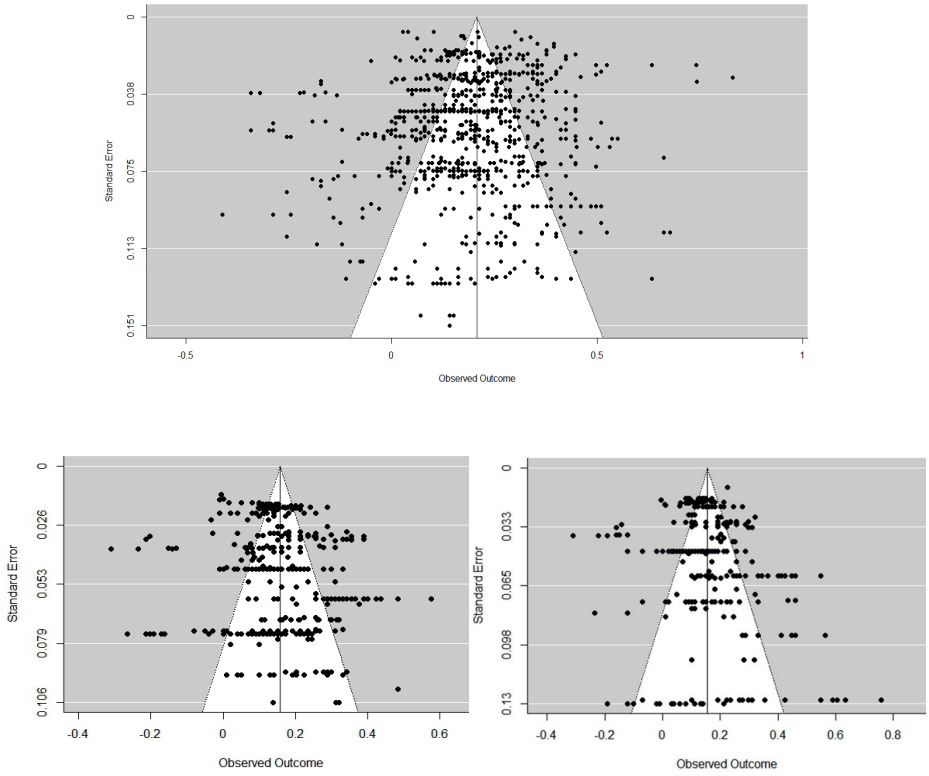

Figure 4. Funnel plots for the associations between parenting and self-control for cross-sectional (A, above), longitudinal parenting → self-control (B, below left), and longitudinal self-control → parenting (C, below right)

Longitudinal P → SC studies

Moderator analyses. Moderator analyses were only conducted if each category contained at least five studies (parameter estimates are poor when number of studies is very small), and summarized in Table 6. As a result, we could not test the moderating effect of adolescent gender, and report informant of self-control. Regarding the moderators of theoretical interest, the only significant moderator was parent gender, $Q_E(366) = 3188.953$, $p < .001$; Omnibus test: $F(2, 366) = 6.150$, $p = .002$. None of the other moderators of theoretical interest, including type of parenting (i.e., positive / negative / relationship), or cultural characteristics (ethnicity, Hofstede's individualism), was significant. Pertaining to the moderators of methodological interest, we tested informant of parenting measure and consistency of informants. Both report informant of parenting measure ($Q_E(360) = 3132.167$, $p < .001$, Omnibus test: $F(3,360)$

= 3.770, p = 0.011), and consistency of informants (Q_E(371) = 3186.385, Omnibus test: F(1,371) = 6.562, p = 0.011), yielded significant results.

Table 6. Longitudinal association P → SC: the Q_E statistics testing residual heterogeneity, and the Omnibus to test the effects of the moderators

Moderator	Q_E(df)	*p*	*Omnibus test*	*p*
Theoretical moderators				
Ethnicity of adolescents	2623.127 (319)	< .001	F(5, 319) = 0.233	.948
Hofstede's individualism	3345.279 (371)	< .001	F(1, 371) = 0.422	.516
Type of Parenting	3243.333 (370)	< .001	F(2, 370) = 1.303	.273
Parent gender	3188.953 (366)	< .001	F(2, 366) = 6.150	.002
Methodological moderators				
Report informant of parenting measure	3132.167 (360)	< .001	F(3, 360) = 3.770	.011
Consistency informants	3186.385 (371)	< .001	F(1, 371) = 6.562	.011

Significant moderators. Follow-up analyses were conducted to analyze the aforementioned significant moderators with results presented in Table 7. Regarding parent gender, we found significant associations for "maternal parenting – self-control", "paternal parenting – self-control", and "both parents' parenting – self-control". Results of further comparison suggested that the association was significantly smaller for the parenting referred to 'both parents' than for parenting referred to either mother or father. The association between parenting and self-control was not significantly different for mothers and fathers.

With respect to the report informant of parenting measures, we found that effect sizes of the relationship between parenting and self-control were all significant when parenting was assessed using adolescent self-report, other-report, observation, and composite measures. Follow-up comparison indicated that effect sizes of studies using composite measures to assess parenting were significantly larger than those using self-report, other-report, and observation. For the consistency of the report informant of parenting and self-control measures, effect sizes were both significant for studies using consistent and inconsistent report informant. Results of follow-up comparison showed that effect sizes of studies using inconsistent report informants were significantly larger than those using a consistent report informant.

Multiple moderator model. For the multiple moderator model, all significant moderators illustrated in Table 8 were included into one single model to test their robustness. The results suggested that at least one of the regression coefficients of the moderators significantly deviated from zero ($F(6,353) = 4.278$, $p < .001$). These results indicated that parent gender (both vs mother), and inconsistent informant reports (vs. consistent) had unique moderating effects on the relationship between parenting and self-control.

Publication bias. The funnel plot is illustrated in Figure 4b, with results of the regression test suggesting significant asymmetry, $z = 2.389$, $p = .017$. This suggests that there was indication of publication bias for longitudinal studies on the association between parenting and self-control. Subsequently, a trim and fill procedure was applied to take publication bias into account, resulting in an adjusted effect size of $ES_z = 0.106$, *S.E.* = 0.007, 95% CI = [0.092, 0.119], $p < .001$, $r = .105$, 95% CI = [.092, .118].

Table 7. Longitudinal associations P → SC: significant moderators

Moderators	***#ES***	***ES_z***	***SE***	***t***	***95% CI (ES_z)***	***p***	***r***
Gender parent	369						
Mother	129	0.194	0.018	10.967	[0.159, 0.229]	<.001	.192
Father	47	0.181	0.023	7.803	[0.135, 0.227]	<.001	.178
Both	193	0.129	0.017	7.590	[0.096, 0.163]	<.001	.129
ΔSlope of mother (vs. father)		-0.013	0.018	-0.705	[-0.048, 0.023]	.481	-.013
ΔSlope of mother (vs. both)		-0.064	0.018	-3.494	[-0.101,-0.028]	<.001	-.060
ΔSlope of father (vs. both)		-0.052	0.024	-2.127	[-0.100 , -0.004]	.034	-.050
Report informant of parenting measure	364						
Adolescent self-report	171	0.152	0.018	8.554	[0.117, 0.187]	<.001	.151
Other-report	62	0.150	0.021	7.013	[0.108, 0.192]	<.001	.149
Observation	44	0.112	0.032	3.538	[0.050 -0.175]	<.001	.110
Composite	87	0.218	0.025	8.881	[0.169, 0.266]	<.001	.215
ΔSlope of adolescent self-report (vs. other-report)		-0.002	0.021	-0.102	[-0.044, 0.040]	.919	.000
ΔSlope of adolescent self-report (vs. observation)		-0.040	0.034	-1.188	[-0.106, 0.026]	.236	.040
ΔSlope of adolescent self-report (vs. composite)		0.065	0.023	2.874	[0.021, 0.110]	.004	.065
ΔSlope of other-report (vs. observation)		-0.038	0.033	-1.159	[-0.102, 0.026]	.247	-.038
ΔSlope of other-report (vs. composite)		0.068	0.028	2.377	[0.012, 0.124]	.018	.068
ΔSlope of observation (vs. composite)		0.105	0.037	2.853	[0.033, 0.178]	.005	-.105
Consistency of report informant	373						
Consistent	155	0.138	0.017	8.259	[0.105, 0.170]	<.001	.137
Inconsistent	218	0.179	0.017	10.610	[0.146, 0.212]	<.001	.177
ΔSlope of consistent report informant (vs. inconsistent)		0.041	0.016	2.562	[0.010, 0.072]	.011	.041

Table 8. Longitudinal association P → SC: Results for the multiple moderator model

Moderator variables	**β (SE)**	**95% CI**	***t***	***p***
Intercept	.179 (.021)	[.138, .221]	8.514	<.001
Gender parent: father (vs. mother)	-.016 (.018)	[-.051,.019]	-0.900	.369
Gender parent: both (vs. mother)	-.061 (.020)	[-.100, -.022]	-3.068	.002
Report informant of parenting measure: other report (vs. self-report)	-.022 (.023)	[-.067, .022]	-0.985	.325
Report informant of parenting measure: observation (vs. self-report)	-.067 (.037)	[-.140,.005]	-1.835	.067
Report informant of parenting measure: composite (vs. self-report)	.030 (.026)	[-.021, .081]	1.461	.253
Consistency of report informant: inconsistent (vs. consistent)	.043 (.020)	[.004, .081]	2.188	.029
Omnibus test	$F(6, 353) = 4.278$, $p < .001$			
Variance within study	.060, $p < .001$			
Variance between studies	.050, $p < .001$			
# ES	360			

Longitudinal SC → P

Moderator analyses. Similar to longitudinal P → SC analyses, we could not test the moderating effect of adolescent gender, and informant on self-control because these categories did not contain enough studies. Results are presented in Table 9. Regarding the moderators of theoretical interest, none of them was significant, including type of parenting (i.e., positive / negative / relationship), cultural characteristics (ethnicity, Hofstede's individualism), and parent gender (maternal parenting / paternal parenting / both parents)

Pertaining to the moderators of methodological interest, including report informant of parenting measure and consistency of informants, only report informant of parenting measure yielded significant results ($Q_E(286)$ = 1999.973, $p < .001$; Omnibus test: $F(3, 286) = 7.075$, $p < .001$).

Table 9. Longitudinal association SC → P: the QE statistics testing residual heterogeneity, and the Omnibus to test the effect of the moderators

Moderator	Q_E (df)	*p*	*Omnibus test*	*p*
Moderators of theoretical interest				
Ethnicity of adolescents	1881.894 (246)	<.001	$F(5, 246) = 1.303$	.263
Hofstede's individualism	2253.226 (289)	<.001	$F(1, 289) = 0.323$	.570
Type of Parenting	2263.721 (288)	<.001	$F(2, 288) = 0.122$	.885
Gender of parents	2264.139 (287)	<.001	$F(2, 287) = 2.354$	.097
Methodological characteristics				
Report informant of parenting measure	1999.973 (286)	<.001	$F(3, 286) = 7.075$	<.001
Consistency informants	2231.286 (289)	<.001	$F(1, 289) = 1.100$	.295

Significant moderators. Follow-up analyses were conducted to further analyze the significant moderators and the results are summarized in Table 10. With respect to the report informant of parenting measure, we found that effect sizes of the relation between self-control and parenting were all significant when parenting was assessed using adolescent self-report, other-report, observation, and composite measures. Follow-up comparison indicated that effect sizes of studies using composite measures to assess parenting were significantly larger than those using self-report, other-report, or observation. Effect sizes of studies using self-report were significantly larger than those using other report informant. No multiple moderator analyses were applied because only one moderator was significant.

Publication bias. Funnel plot (illustrated in Figure 4c.) and regression test indicated publication bias, $z = 3.694$, $p < .001$. Accordingly, trim and fill procedure was applied to take publication bias into account, resulting in an adjusted effect size of $ES_z = 0.153$, *S.E.* = 0.007, 95% CI = [0.139, 0.167], $p < .001$, $r = .152$, 95% CI = [.138, .165].

Summary of results

In order to let readers have a straightforward view of the results, we have summarized the overall cross-sectional and longitudinal associations and the significance of theoretical and methodological moderators in Table 11.

Table 10. Longitudinal associations SC → P : significant moderators

Moderators	**#ES**	***ES$_z$***	***SE***	***t***	***95% CI (ES$_z$)***	***p***	***r***
Report informant of parenting measure							
Self-report	128	0.154	0.024	6.478	[0.107, 0.201]	<.001	.153
Other report	34	0.096	0.026	3.626	[0.044, 0.148]	<.001	.096
Observation	37	0.113	0.034	3.331	[0.046, 0.180]	<.001	.113
Composite	91	0.232	0.035	6.684	[0.164, 0.300]	<.001	.228
ΔSlope self-report (vs. other report)		-0.058	0.018	-3.303	[-0.093, -0.024]	.001	-.058
ΔSlope self-report (vs. observation)		-0.041	0.036	-1.130	[-0.112, 0.030]	.260	-.041
ΔSlope self-report (vs. composite)		0.078	0.039	1.994	[0.001, 0.154]	.047	.078
ΔSlope other-report (vs. observation)		0.018	0.037	0.478	[-0.055, 0.090]	.633	.018
ΔSlope other-report (vs. composite)		0.136	0.040	3.418	[0.058, 0.214]	<.001	.135
ΔSlope observation (vs. composite)		0.118	0.038	3.113	[0.044, 0.193]	.002	.117
Omnibus test	$F(3, 286) = 7.075$, $p < .001$						
Variance within study	.040, $p < .001$						
Variance between studies	.100, $p < .001$						
# ES	290						

Table 11. Summary of overall effects and results of moderation analyses based on multiple moderator tests

	Cross-sectional	**Longitudinal P → SC**	**Longitudinal SC → P**
Overall association (r)	0.204	0.157	0.155
Theoretical moderators			
Ethnicity of adolescents	not significant	not significant	not significant
Hofstede's individualism	not significant	not significant	not significant
Type of parenting	positive > negative parenting	not significant	not significant
Parent gender	not significant	'both parents' assessed < mother	not significant
Adolescent gender	not significant	not enough information	not enough information
Age	not significant	not applicable	not applicable
Methodological moderators			
Report informant of parenting measure	not significant	not significant	composite effect size > other three informants
Report informant of self-control measure	composite > self-report	not enough information	not enough information
Consistency in informants	consistent > inconsistent	inconsistent > consistent	not significant

Table 12. Recommendation of sample size

	α =.05; power =.80; two-tailed	**α =.05; power =.90; two-tailed**	**α =.05; power =.95; two-tailed**	**α =.05; power =.99; two-tailed**
Cross-sectional design (*r* = .204)	186	248	306	432
Longitudinal design: parenting → self-control (*r* = .157)	316	422	521	736
Longitudinal design:self-control → parenting (*r* = .155)	324	433	535	755

Note. Software version: G*Power 3.1.9.2; Test family: exact; Statistical test: correlation: bivariate normal model.

Auxiliary analyses

Effect size for the association between specific parenting dimensions and self-control. While we subcategorized our parenting dimensions into the categories positive, negative and relationships, we realize that the investigation of subcategories of parenting presented in Table 1 could be of interest to the field. We therefore provide free online access to our data, with details on specific parenting dimensions and analytic scripts. These data include detailed explanations so that researchers can easily add their data and/or further analyze the association between specific parenting dimensions and self-control and potential moderators influencing this association. Exploratory analyses between specific parenting dimensions (e.g., authoritative, supervision, negative control, and attachment) and self-control from cross-sectional and longitudinal associations are presented in the supplement Table 1. As shown in the table, effect sizes of most subcategories of parenting were significantly related to self-control with few exceptions (e.g., longitudinal association from positive expressivity / conflict to self-control). In addition, the magnitudes of these effect sizes vary to some extent. For instance, *relationship* is the parenting subcategories related to self-control with largest effect sizes in both cross-sectional and longitudinal associations, while *harshness* and *conflict* had smallest effect size for cross-sectional and longitudinal associations, respectively. For the longitudinal effect of self-control on parenting, *relationship* and *harshness* are most and least likely to be affected by adolescents' previous levels of self-control, respectively.

Power analysis. Table 12 displays the sample size needed for detecting the average correlation found by this study at different levels of statistical power. For instance, with .05 as significant level and .204 as coefficient in cross-sectional study, approximately 186 participants are needed to achieve .80 statistical power, 248 participants for .90 statistical power, 306 participants for .95 statistical power, and 432 for .99 statistical power.

DISCUSSION

Parenting has long been considered to be important to the development of self-control (Gottfredson & Hirschi, 1990; Eisenberg et al., 2003, 2005; Kochanska et al., 1995; Kopp, 1982; Sameroff, 2010). The current three-level meta-analysis is the first to synthesize the relation between broad types of parenting (i.e., positive parenting, negative parenting, and parent-adolescent

relationship) and self-control of adolescents aged 10 to 22 years. Analyses were based on 191 studies, 1,540 effect sizes, from four continents, with a sample size of *N*=164,459. Our results showed that: (1) parenting is concurrently and longitudinally associated with self-control throughout adolescence; (2) adolescent self-control significantly predicts subsequent parenting and the predictive effect of parenting on self-control and the one of self-control on parenting show similar magnitude; and (3) the relations between parenting and self-control (for both directions) largely hold equal across cultures, ethnicities, parent and adolescent gender, and age, while the associations are moderated by a few methodological factors, such as report informant consistency. These findings provide a rich description of whether and how self-control and parenting are related across the entire period of adolescence.

Despite the changes that occur in adolescence such as eminent independence from parents and more investment in peer and romantic relationships (Connolly et al., 2004; Nickerson & Nagle, 2005; Song et al., 2009), our results showed that parenting was related to self-control in adolescence both concurrently and longitudinally. Speaking to the robustness of our findings, the effect sizes for the "parent-child relationship – self-control" association were similar to those reported in a recent meta-analysis focusing on the attachment security status and its relation with effortful control in children and adolescents up to age 18 (Pallini et al., 2018).

Beyond the influence of parenting on self-control, our findings based on longitudinal studies also revealed a significant effect of adolescent self-control on subsequent parenting. Previous research has assumed an evocative effect from child's outcomes to parenting (e.g., Kochanska et al., 1995; Silverman & Ragusa, 1990; Sameroff, 2010), but existing findings were inconsistent (e.g., Lee et al., 2012; Meldrum et al., 2012; Moilanen et al., 2015). The present findings, based on 291 effect sizes, provide support for the notion that adolescent self-control at a given time point does affect subsequent parenting behaviors, such that high self-control leads to more positive parenting, less negative parenting, and a better positive parent-adolescent relationship. Low self-control, in contrast, is linked to diminished use of positive parenting (e.g., warmth, support) and increased use of negative parenting (e.g., physical and coercive punishment), and gives rise to a more negative parent-child relationship. We did not find a significant difference in the magnitude of the effect of parenting on adolescent self-control and the effect of adolescent self-control on parenting, which is consistent with developmental theories underpinning the importance of bidirectional interactions between adolescents

and their (parental) environment for adolescent development (Bandura, 1999; Bronfenbrenner, 1979; Sameroff, 2010; Plomin et al., 1977). The current results revealed that, throughout adolescence, parenting continues to affect the development of adolescent self-control and, adolescent self-control continues to affect parenting.

The associations tested were found to be moderated by a few—mainly methodological—factors (see summary in Table 11). However, moderators for cross-sectional studies did not necessarily extend to longitudinal studies (e.g., type of parenting) and vice versa (e.g., parent gender). Moreover, for some moderators, there was not enough information to detect their effects (e.g., adolescent gender for longitudinal studies). Some moderators (e.g., consistency of report informants) even showed contradictory moderating effects for cross-sectional and longitudinal studies. For example, in cross-sectional studies effect sizes were larger for studies using consistent rather than inconsistent report informants, but in longitudinal studies (SC → P) effect sizes were larger for studies using inconsistent rather than consistent report informants.

Based on cross-sectional studies, which comprised more than half of the total effect sizes, we found that the relation between parenting and self-control tended to be stronger when parenting was positive than when parenting was negative, when self-control was measured using multiple report-informants compared to single report informant, and when informants of parenting and self-control were consistent.

Given the small moderating effects, our results suggest that the inconsistent findings regarding the association between parenting and self-control in the past literature may be largely due to methodological artifacts rather than theoretical misspecification. In addition, we found a publication bias in longitudinal but not in cross-sectional studies, which may be a reason why moderators between cross-sectional and longitudinal studies were not entirely consistent, because publication bias may also influence the estimates of between-study variance (Jackson, 2006).

Theoretical implications

The present results bear several theoretical implications. First, a number of theories and theoretical perspectives (e.g., attachment theory, the general theory of crime, the development of self-control and conscience; and the unified theory of development) propose that good parenting is a crucial source of self-control in children (Bowlby, 1969; Gottfredson & Hirschi, 1990; Kochanska, 1993; Kopp, 1982; Sameroff, 2010). The current findings imply

that this proposition extends to adolescence, and show that positive parenting and good parent-child relationships continue to play an essential role in shaping the development of self-control from early to late adolescence; conversely, negative parenting and poor parent-child relationship continue to hamper the development of adolescents' self-control. Second, the importance of parenting on adolescent self-control is largely equivalent across different cultures, ethnicities, and adolescent and parent gender. This suggests that the above mentioned theories and viewpoints regarding the influence of parenting on self-control are generally applicable across different demographic backgrounds, thus demonstrating their cross-cultural validity. Third, the aforesaid theories and perspectives mainly focus on parental effect on adolescents' self-control and disregard the examination of the evocative effect, as pointed out by some scholars (Lerner, 2002; Tiberio et al., 2016; Vazsonyi & Huang, 2010). The current results demonstrated both parent- and child-effect regarding the relations between parenting and self-control, which suggests that the existing theories may need to take both parent and child effects into consideration to better capture the dynamic relation between parenting and self-control in adolescents.

Limitations

The present findings should be interpreted with caution. First, the sample size for the cross-sectional analyses was much larger than the sample size for the longitudinal analyses, resulting in more powerful analyses for the former. Due to this power issue, not all moderators could be tested in the longitudinal analyses.

Second, our meta-analysis only included community-based adolescent samples, and its results may not be generalized to clinical samples (e.g., diagnosed with attentional disorder and hyperactivity disorder, autism spectrum disorder, diabetes) or samples with specific characteristics proposed to be related to self-control (e.g., prisoners, drug-addicts).

Third, we acknowledge that the results based on the longitudinal studies refer to lagged associations/effects but not to *changes* in self-control or parenting since we did not control for the baseline levels of these constructs for two reasons. Conceptually, most longitudinal studies not only control for baseline levels of self-control or parenting, they also control for other covariates (e.g., child gender, age, etc.). This makes the beta coefficients non-comparable across studies. Methodologically, although we are aware that some new techniques such as metaSEM (Cheung, 2015) have the potentials to control for the target

construct in meta-analysis, the current version of such techniques is not as able to deal with dependency problems as multi-level meta-analysis (the one applied in the present study). Not dealing with dependency problem possibly loses much information, reduces statistical power and even leads to bias results (Hox et al., 2010; van den Noortgate et al., 2013). Nevertheless, we encourage scholars in the future to revisit this issue when more sophisticated statistical approaches evolve.

Last, the "similar effect size" for the longitudinal effect of parenting on adolescent self-control and the one of adolescent self-control on parenting refers to the effect sizes *before* adjusting for publication bias. After taking publication bias into account, the magnitudes of the two effect sizes appeared different. However, the trim-and-fill approach is controversial as it imputes non-existing effect sizes (Sutton et al., 2000), and effect sizes as a result of such analyses cannot be convincingly compared. Therefore, this result should be interpreted with caution and take publication bias and the limitation of the trim-and-fill approach into consideration.

Future directions

The current study bears important implications for future research. First, the included studies came from several continents and the sample size was large (over 160,000). However, a majority of effect sizes (i.e., 62%) were retrieved from studies conducted in the USA, and no or few eligible effect sizes were based on studies from African, South American, Southeastern Asian, Central Asian, and Eastern European countries. In order to further strengthen the current findings, we encourage scholars to integrate findings published in a wider variety of languages into our open access dataset. Doing so will eventually accumulate more effect sizes from a more varied population, which allows scholars to test a wider range of moderators and to achieve results with greater generalizability and higher robustness. Considering the fact that our data and scripts are freely accessible online, extending our results with international data is feasible. This also provides opportunities for other scholars who have different theoretical preference to categorize parenting such as warmth / behavioral control / autonomy support (e.g., Prinzie, Stams, Deković, Reijntjes, & Belsky, 2009) to analyze our data for different research questions and facilitates an update of the meta-analysis in the future.

Second, among the studies included in this meta-analysis, many assessed parenting in general without separately referring to mothers or fathers. Although mothers' parenting and fathers' parenting often show medium-to-

high correlations (e.g., Li et al., 2015; Ng-Knight, Shelton, Frederickson, McManus, & Rice, 2018; Nie et al., 2016; Özdemir et al., 2013), examining parenting for mothers and fathers separately would be promising. It may allow researchers to identify the similarities and differences between maternal and paternal influences on adolescent self-control. Future research regarding this issue may want to separately explore the effect of mothers and fathers.

Third, the magnitude of the effect sizes suggests that adolescent self-control is influenced by multiple socialization agents. For example, peers and teachers are also potential socializing agents steering adolescents' self-control (e.g., Alvarez-Rivera & Fox, 2010; Meldrum, 2008; Turner, Piquero, & Pratt, 2005). Importantly, a recent meta-analysis shows that the heritability of self-control is 60%, highlighting that individual differences in self-control are not only the result of socializing factors but also the result of biological factors (Willems, Boesen, Li, Finkenauer, & Bartels, 2019). However, much of the literature to date evaluates the development of self-control as a result of environmental socialization. Incorporation of biological studies is necessary to paint a more complete picture of individual differences in self-control. Future studies applying genetically sensitive designs are particularly promising, as these allow researchers to investigate whether the association between parenting and self-control is genetically based, environmentally based or a combination of these (Willems et al., 2019b).

Last, this meta-analysis provided overall effect sizes for cross-sectional and longitudinal associations between parenting and self-control. This knowledge on the average effect size allows us to provide additional recommendations for future research. Specifically, it provides information on the number of participants necessary to detect the current findings. Doing so will allow researchers to gauge the appropriate level of conservatism or liberalism they prefer when recruiting participants, and helps researchers to make the most of their time and resources. Table 12 summarizes sample sizes to achieve the correlation coefficients transformed back from effect sizes at four levels of power with alpha level of .05. It should be noted that these sample sizes are estimated for bivariate correlations. If researchers wish to conduct other statistical analyses in future studies, they may need to recalculate the sample size based on the effect sizes found in this study. However, this can be easily implemented in G*Power Software or using other approaches (e.g., Monte Carlo simulation).

Conclusive remarks

Ill decisions and reckless behaviors due to low self-control in adolescence are at the cost of individual physical and psychosocial functioning as well as social security, both concurrently and longitudinally (Moffit et al., 2011; Caspi et al., 2017). The current study suggests that parenting significantly contributes to self-control in adolescents aged 10 to 22. It also suggests that adolescent self-control shows a significant lagged effect on subsequent parenting. These relationships are largely equal across cultures, ethnicities, parent and child gender, and age of adolescents; and only a few (mainly methodological) factors moderate this relationship. Our findings provide further evidence for the importance of considering the continuous and dynamic interplay of the development of self-control and environment (parenting/parent-child relationship) across the adolescent period.

SUPPLEMENTS

CHAPTER 2

Supplemental Table 1. Associations between specific dimensions of parenting and self-control for cross-sectional and longitudinal effect sizes

Specific parenting dimensions	*#ES*	*ES_z*	*SE*	*t*	*95% CI(ES_z)*	*p*	*r*
Cross-sectional associations							
Authoritative	66	.183	.025	7.227	[.133, .233]	<.001	.181
Monitoring	69	.180	.019	9.434	[.143, .218]	<.001	.178
Support	98	.243	.018	13.416	[.208, .279]	<.001	.238
Responsive discipline	36	.255	.026	9.895	[.204, .306]	<.001	.250
Warmth	50	.210	.023	9.141	[.165, .255]	<.001	.207
Supervision	16	.180	.036	4.997	[.109, .250]	<.001	.178
Positive expressivity	7	.217	.059	3.679	[.101, .333]	<.001	.214
Conflict	50	.234	.028	8.453	[.180, .289]	<.001	.230
Authoritarian	55	.185	.027	6.931	[.133, .238]	<.001	.183
Negative control	66	.157	.021	7.501	[.116, .198]	<.001	.156
Harshness	30	.106	.030	3.508	[.047, .165]	<.001	.106
Permissive	32	.151	.028	5.310	[.095, .207]	<.001	.150
Attachment	64	.221	.026	9.355	[.190, .291]	<.001	.217
Relationship	31	.273	.029	9.355	[.216, .330]	<.001	.266
Longitudinal associations P → SC							
Authoritative	36	.161	.029	5.617	[.105, .217]	<.001	.160
Monitoring	27	.186	.031	6.017	[.125, .246]	<.001	.183
Support	24	.083	.033	2.543	[.019, .147]	.012	.083
Responsive discipline	34	.218	.025	8.631	[.168, .268]	<.001	.215
Warmth	17	.164	.038	4.359	[.090, .238]	<.001	.163
Supervision	22	.148	.033	4.531	[.084, 213]	<.001	.147
Positive expressivity	9	.097	.055	1.771	[-.011, .205]	.078	.100
Conflict	35	.060	.081	0.743	[-.099, .219]	.458	.060
Authoritarian	20	.200	.041	4.842	[.118, .281]	<.001	.197
Negative control	6	.124	.041	3.031	[.043, .204]	.003	.123
Harshness	26	.107	.032	3.292	[.043, .171]	.001	.107
Permissive	6	.130	.044	2.926	[.043, .218]	.004	.130
Attachment	27	.156	.030	5.214	[.097, .215]	<.001	.154
Relationship	9	.264	.035	7.546	[.195, .333]	<.001	.258

Longitudinal associations SC → P							
Authoritative	37	.189	.035	5.417	[.120, .258]	<.001	.187
Monitoring	41	.131	.034	3.800	[.063, .199]	<.001	.130
Support	11	.247	.048	5.173	[.153, .341]	<.001	.242
Responsive discipline	25	.288	.036	8.053	[.217, .358]	<.001	.280
Warmth	14	.143	.070	2.051	[.006, .280]	.041	.142
Supervision	15	.192	.055	3.484	[.083, .300]	<.001	.190
Positive expressivity	9	.135	.077	1.747	[-.017, .287]	.082	.134
Conflict	32	.191	.129	1.475	[-.064, .446]	.141	.189
Authoritarian	22	.247	.037	6.715	[.174, .319]	<.001	.242
Negative control	9	.180	.039	4.597	[.103, .258]	<.001	.178
Harshness	22	.010	.044	.218	[-.077, .097]	.828	.010
Permissive	6	.235	.043	5.512	[.151, .320]	<.001	.231
Attachment	17	.180	.053	3.412	[.076, .284]	<.001	.178
Relationship	7	.299	.046	6.510	[.209, .390]	<.001	.290

Note: P = parenting; SC = self-control. Only parenting dimensions with no less than 5 effect sizes in all the three categories are included to assure statistical power. To ensure similar direction of effects, effect sizes were recoded if 1) parenting pertained to negative dimensions, and 2) self-control was measured using a scale of 'lack of self-control', or 'low self-control'. As such, for the negative parenting dimensions correlations appear positive, but reflect a negative correlation between negative parenting and self-control. The 95% CI (*r*) was calculated using fisher-z-to-r formula based on lower and upper bound of the 95% CI (ES_z).

Chapter 3

The relationship between family violence and self-control in adolescence: A multi-level meta-analysis

ABSTRACT

Theoretical studies propose an association between family violence and low self-control in adolescence; however, empirical findings of this association are inconclusive. The aim of the present research was to systematically summarize available findings on the relation between family violence and self-control across adolescence. We included 28 studies with 143 effect sizes, representing more than 25,000 participants of eight countries from early to late adolescence. Applying a three-level meta-analysis, taking dependency between effect sizes into account while retaining statistical power, we examined the magnitude and direction of the overall effect size. Additionally, we investigated whether theoretical moderators (e.g., age, gender, country), and methodological moderators (e.g., time lag between family violence and self-control, informant) influenced the magnitude of the association between family violence and self-control. Our results revealed that family violence and self-control have a small to moderate significant negative association (r = −0.191). This association did not vary across gender, country, and informants. The strength of the association, however, decreased with age and in longitudinal studies. This finding provides evidence that researchers and clinicians may expect low self-control in the wake of family violence, especially in early adolescence. Recommendations for future research in the area are discussed.

Keywords: family violence; self-control; meta-analysis; adolescence

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Family violence—relational escalations in which one or more family members engage in verbal or physical violence—is common and has tremendous costs for individuals, communities and society. Individuals exposed to family violence show increased vulnerability to decrements in physical, mental, and social wellbeing across the lifespan (Kessler et al., 2007; Patton et al., 2014). It is a particularly harmful risk factor during adolescence, as family violence may jeopardize not only adolescents' current wellbeing, but also their wellbeing as adults, and even the wellbeing of their future children (Buehler et al., 1997; Habib et al., 2014; Repetti, Taylor, & Seeman, 2002). Importantly, experiencing family violence predicts adolescents' use of violence themselves, generating a vicious circle of violence from one generation to the next (Cui, Durtschi, Donnellan, Lorenz, & Conger, 2010; Ehrensaft et al., 2003). Although there is a consistent link between family violence and adverse outcomes for adolescents, development of effective prevention and intervention strategies would benefit from more knowledge on the specific processes underlying this link.

Recent theoretical studies propose that self-control plays a key role in the family violence—adverse outcome link because of its foundational function in regulating behavior, emotions, and cognition (Finkenauer et al., 2018; Finkenauer et al., 2015). Family violence may decrease adolescents' self-control, and this decrease, in turn, is likely to carry over to cause adverse outcomes in other domains such as school, with peers, and in romantic relationships. Moreover, lowered self-control as a result of repeated exposure to family violence could make adolescents more likely lose self-control in stressful situations (DeWall, Finkel, & Denson, 2011), thereby exacerbating violence within their family. Empirical evidence of these two theoretical core propositions, however, has produced mixed results. To illustrate, some studies find a significant association (Agbaria, Hamama, Orkibi, Gabriel-Fried, & Ronen, 2016), while others show support for a cross-sectional and a longitudinal link between family violence and low adolescent self-control (Hallquist, Hipwell, & Stepp, 2015), and again other studies find a cross-sectional but not a longitudinal association (Moilanen, Rasmussen, & Padilla-Walker, 2015; Park & Kim, 2012), or find an effect from low self-control to family violence but no evidence for the reverse relation (Brody & Ge, 2001; Caprara, Gerbino, Paciello, Di Giunta, & Pastorelli, 2010). To shed light on the relation between family violence and self-control, this paper aims to summarize and quantify the association between family violence and self-control across adolescence through applying a three-level meta-analysis.

Conceptualization of self-control

Self-control involves the ability to initiate desirable actions and behaviors (e.g., finish homework, concentrate in class, achieve goals), and the capacity to inhibit undesirable impulses (e.g., suppress procrastination, overcome temper tantrums, avoid rule breaking; (de Ridder, Lensvelt-Mulders, Finkenauer, Stok, & Baumeister, 2012; Duckworth & Steinberg, 2015). Self-control is an important concept within diverse research traditions, with criminologists and social psychologists embracing the term self-control, developmental psychologists using the terms effortful control, and clinical psychologists preferring the term self-regulation (Duckworth & Steinberg, 2015). Empirical research shows that these terms collectively tap into the capacity to alter unwanted impulses and behavior and bring them into agreement with standards (Allan & Lonigan, 2014; Duckworth & Kern, 2011; Fan, Flombaum, McCandliss, Thomas, & Posner, 2003; Nigg, 2017; Vazsonyi, Mikuška, & Kelley, 2017).

The capacity to perform self-control is of specific importance to adolescents. The teenage years are marked by a range of normative biological and social challenges (Crone & Dahl, 2012), including increases in risk-taking behavior (Boyer, 2006), and social reward seeking (Smetana, Campione-Barr, & Metzger, 2006). Low self-control hinders adolescents' capacity to deal with these challenges. For example, adolescents with low self-control are less happy, have more negative social interactions, perform worse in school, and are more likely to get involved in oppositional behaviors and substance use than adolescents with high self-control (Duckworth, Gendler, & Gross, 2014; Finkenauer, Engels, & Baumeister, 2005; Laceulle, Veenstra, Vollebergh, & Ormel, 2017; Moffitt et al., 2011). Together, these findings highlight the importance of self-control during adolescence for healthy development across the lifespan.

The relationship between family violence and self-control

Family violence is defined as destructive conflict within the family that is violent, frequent, and harmful. We conceptualize family violence as conflict that is frequent, involves verbal and/or physical overt aggression, and conflict that is rancorous or hostile in form and content and comprises multiple family members (Harold & Sellers, 2018; Rhoades, 2008). There are different pathways by which family violence may affect self-control. Family violence induces emotional stress in adolescents, resulting in behavioral, physiological, and cognitive dysregulation and lower self-control (Bridgett, Burt, Edwards, & Deater-Deckard, 2015; Davies & Cummings, 1994; El-Sheikh, Tu, Erath, &

Buckhalt, 2014). Additionally, studies show that family violence is a strong predictor of sleep problems, which, in turn, predicts self-control problems (Baumeister, Vohs, & Tice, 2007; El-Sheikh et al., 2014; Meldrum, Barnes, & Hay, 2015). Rumination as a result of violent interaction is also likely to reduce self-control (Denson, Pedersen, Friese, Hahm, & Roberts, 2011).

Moreover, studies suggest that family violence decreases self-control indirectly through processes associated with the family or the household. For example, family violence is predictive of more harsh discipline and less parental warmth and acceptance, limiting adolescents' opportunities to learn through social observation how to manage their impulses and emotions (Harold & Sellers, 2018; Krishnakumar & Buehler, 2000). Similarly, in families with family violence studies report lower parent-child relationship quality and lower sibling relationship quality which, in turn, undermines adolescents' ability to develop self-controlled behavior (Brody, Stoneman, Smith, & Gibson, 1999; Davies, Sturge-Apple, Cicchetti, Manning, & Zale, 2009; Deković, 1999). These findings are consistent with the suggestion that family violence is negatively related to adolescents' self-control at the within person level (stress, sleep, rumination) and through processes associated with the family and living conditions (parenting, family relationships).

Adolescents, nonetheless, are not passive recipients of their environment and some recent research suggests that adolescents with low self-control may evoke or maintain violence within the family. Adolescents with low self-control are more likely to undermine parental rules, which spurs parents to show over-controlling or hostile parenting strategies, exacerbating violence within the family (Wiener, Biondic, Grimbos, & Herbert, 2016). This is in line with the behavior genetic literature, indicating that genetically influenced traits such as low self-control evoke harsh parenting responses, emphasizing the importance of taking child-driven effects into account (Harold, Leve, & Sellers, 2017; Plomin & Daniels, 2011). Additionally, adolescents with low self-control are considered as less trustworthy by their family members and are less successful in de-escalating conflict (Righetti & Finkenauer, 2011; Vohs & Faber, 2007). Also, individuals with low self-control are more likely to show aggressive behavior in close relationships (Finkel, DeWall, Slotter, Oaten, & Foshee, 2009; Payne, Higgins, & Blackwell, 2010). As such, the association between family violence and self-control can be understood as a transactional or reciprocal process, where contextual factors (family violence) affect the development of adolescents (self-control), and adolescents' behavior evokes or maintains the context in which they develop.

In sum, in order to better understand the association between family violence and self-control, it is important to investigate the magnitude and the directional effect from family context to adolescent and from adolescent to family context (Chapple, Tyler, & Bersani, 2005; Finkenauer et al., 2018; Finkenauer et al., 2015). A meta-analysis including longitudinal studies allows researchers to pit these effects against each other. Longitudinal studies include (a) an effect size where family violence is measured at one time point and self-control is measured at a succeeding time point and/or, (b) an effect size where self-control is measured at one time point and family violence at a succeeding time point, (c) or both. A meta-analysis allows to examine the average magnitude of these different effect sizes respectively.

Moderators of the link between family violence and self-control

An additional key strength of a meta-analysis is that it allows researchers to examine potential boundary conditions under which the relation between family violence and self-control may vary in magnitude. The association may vary as a function of theoretical moderators, such as age, gender, or country, and as a function of methodological moderators, such as whether the correlation pertains to cross-sectional assessments or longitudinal assessments, or to the type of informant.

Theoretical moderators

Age. Research shows that youth of all ages are adversely affected by family violence, yet the magnitude of the effect may vary across adolescence (Harold & Sellers, 2018). Throughout adolescence, teenagers increasingly claim more autonomy. As a result, some researchers argue that the association between family violence and low self-control is stronger during early adolescence, when teenagers are on the verge of gaining independence but still rely on parental support, than in later adolescence, when other social contexts and socializing agents become increasingly important (e.g., peers, school, neighborhood, Sameroff, 2010). Other evidence, however, suggests that the association increases over the course of adolescence because older children are likely to have been exposed to violence for a longer period of time (Pinquart, 2017; Rhoades, 2008). Accordingly, in this meta-analysis we will explore whether the association between family violence and self-control changes as adolescents grow older.

Adolescent gender. Evidence suggests that the effects of family violence are equally harmful for boys and girls (Harold & Sellers, 2018). Differences between boys and girls do become apparent in the way they perceive family violence;

boys are more likely to perceive violence as a personal threat, while girls are more likely to perceive it as a threat to the harmony of the family system (Davies & Lindsay, 2004). As a result, some research suggests gender differences in the developmental trajectories of the association between family violence and self-control. Specifically, research found that for girls the association was stronger during adolescence while for boys it was stronger in early childhood (Davies & Lindsay, 2004). This study will explore whether the association between family violence and self-control is moderated by adolescent gender.

Country. In their discussion sections, studies on the family violence—self-control link often suggest that findings should be replicated in different populations and international contexts. While we do not have specific hypothesis regarding country effects, a meta-analysis allows us to explore whether the association between family violence and self-control varies across countries or cultures. Moving beyond the classical "West" versus "East" paradigm, existing meta-analyses apply the continuous and nuanced culture scores developed by Hofstede to examine differences between countries (Hofstede & McCrae, 2004; Parks-Leduc, Feldman, & Bardi, 2015; Piotrowska, Stride, Croft, & Rowe, 2015). These scores allow researchers to rate countries according to their level of individualism, attitude towards unequal distribution of power, and focus on competition and achievement within society. Applying such scores as moderators allows us to explore whether the association between family violence and self-control is generalizable across countries, or whether it shows different patterns across cultural dimensions (Li, Delvecchio, Lis, Nie, & Di Riso, 2015; Ng, Pomerantz, & Deng, 2014; Smetana, 2017; Vazsonyi, Trejos-Castillo, & Huang, 2006).

Methodological moderators

Time lag between family violence and self-control. Studies investigating the association between family violence and self-control have applied concurrent and/or prospective study designs: some assessed a cross-sectional association between family violence and self-control whereas others examined a longitudinal association. The differences in the magnitude of cross-sectional versus longitudinal studies are, however, not well quantified. An earlier meta-analyses on the link between attachment and self-control across the lifespan found larger effect sizes for cross-sectional studies as compared to longitudinal studies (Pallini et al., 2018). In the same vein, this meta-analysis will explore whether the magnitude of the association between family violence and self-control differs with the time lag between family violence and self-control.

Informants. The magnitude of the association between family violence and self-control could vary depending on methodological specifications, such as the way violence and self-control are assessed (e.g., parent report or adolescent self-report), and whether they are assessed by the same informant (e.g., both self-report or both parent report, (Duckworth & Kern, 2011). For self-control, correlations between self-reports are on average stronger than correlations between self-reports and other reports (Willems et al., 2018a). As such, we explore whether the association between family violence and self-control differs depending on informant, and whether it differs when both are assessed by the same person.

The present study

While there is evidence for the link between family violence and self-control, empirical evidence regarding the magnitude and the direction of the effect remains inconclusive. The aim of the present study is to 'take stock' of the published literature so far by applying a three-level meta-analysis. A meta-analysis is ideal to summarize the published literature, because it allows for aggregating diverse individual study results to identify the overall mean effect and investigate the role of possible moderators on the magnitude of this effect. Doing so allows us to (1) quantify the relationship between family violence and self-control across adolescence, (2) examine the influence of theoretical and methodological moderators, and (3) elucidate gaps and questions that require attention in future research.

METHOD

Literature search

We collected data through systematic database search of *ERIC*, *PsycInfo*, *Pubmed*, and *Web of Science* until September 2018, following the Preferred Reporting Items for Meta-Analyses (PRISMA) checklist. Search terms included family variables (parent* or mother* or father* or parental or maternal* or attachment* or family* or bond*), self-control variables (self-control or self-regulation or self-discipline or effortful-control), and adolescent variables (adolescent* or adolescence or teen* or youth* or child* or student* or undergraduate or emerging adult* or young adult*). We chose the adolescent age span from age 10 to 22 years, in order to capture the broad developmental range of teenage development (Crone & Dahl, 2012).

In order to ensure extensive search outcomes, we applied search terms capturing broad family variables. First, when reporting on family violence, it is common to mention a family related keyword in the title or abstract (e.g., parent, adolescent, family). In our search, we included all studies that mentioned family related key words in the title or abstract for full text screening (e.g., parent, mother, father, parental, family, bond, adolescent, child). Second, in some studies family violence is not the key focus but included for exploratory analyses. By applying these broad terms, we were able to include studies that specifically focus on the family-self-control association and capture studies that have a different research question but include violence as an explorative variable or covariate. Third, some studies do not explicitly mention family violence in their abstract but apply measures assessing family violence (for example as a dimension of harsh parenting). Our extensive search allowed us to include a large number of studies and inspecting parenting measures thoroughly to detect studies including effect sizes on family violence and self-control.

Studies were included if (1) the study included a correlation between family violence (on any relational level) and self-control, (2) the study included non-clinical samples, (3) the study was published in English, in a peer-reviewed journal, and (4) the age of the participating adolescents was between 10 and 22 years (see Figure 1 for the flowchart).

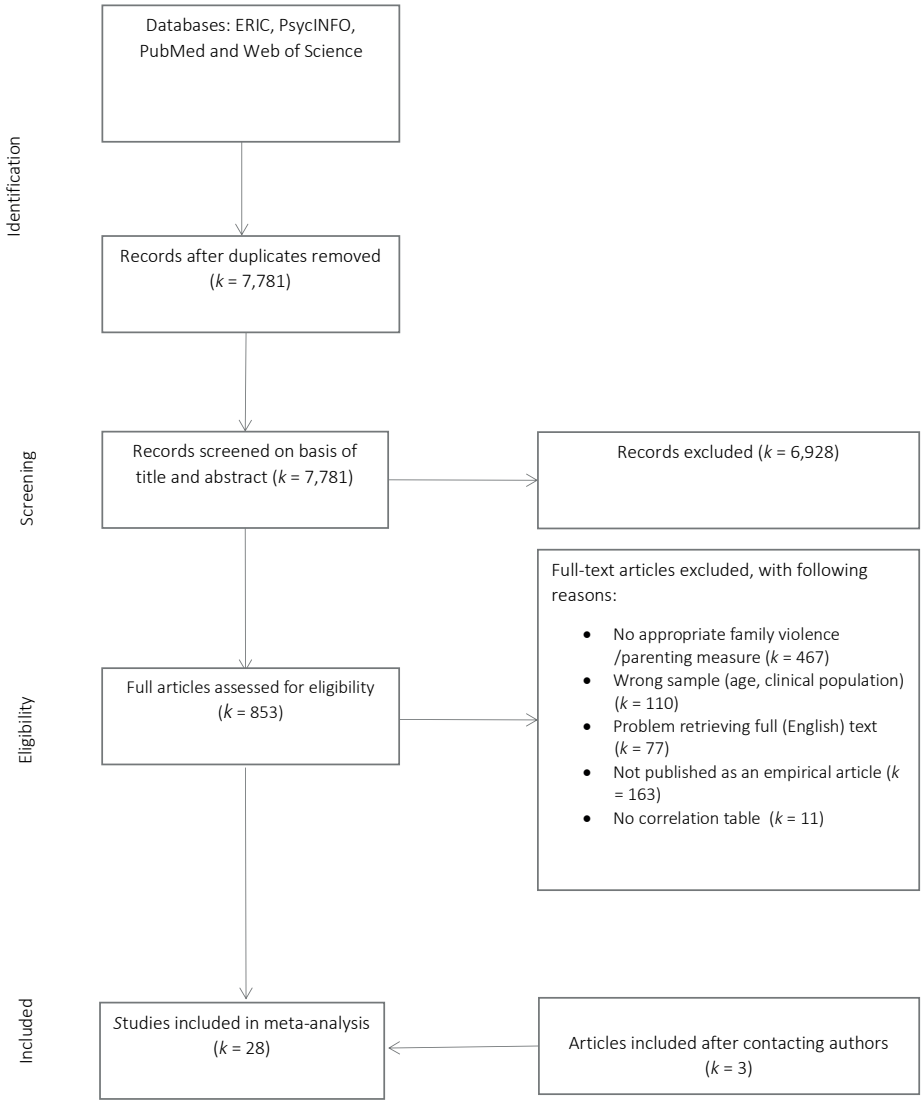

Figure 1. PRISMA flowchart used to identify studies for detailed analysis of parenting and self-control.

Selection of studies

Our search yielded 7781 hits which, after removing duplicates of the multiple search engines and applying inclusion criteria to the title and abstract, resulted in 853 potentially relevant articles for full text screening. Of the 853 articles, k = 467 articles were excluded because they did not measure family violence or self-control (e.g., only mentioned the concept in their introduction or discussion without empirical assessment), k = 110 were excluded because they focused either on the wrong age or on a clinical population, k = 77 were excluded because the full text was not published in English, and k = 163 were excluded because they were not published as an empirical article (e.g., dissertations, student theses, or conference abstracts). Additionally, k = 11 articles did assess the association between family violence and self-control but did not include a correlation table. Authors of these articles were contacted, resulting in an additional k = 3 articles to include in the present meta-analysis. In total, 28 studies met the abovementioned inclusion criteria and were included in the present meta-analysis (see Figure 1 for the flowchart).

We collected relevant information of the studies and organized them according to a detailed coding scheme (Lipsey & Wilson, 2001). This coding scheme included study descriptors (e.g., author names, title, year of publication, data collection details, sample size), moderator variables (e.g., time lag between family violence and self-control, age, country, informant), and the correlation between family violence and self-control (retrieved from correlation tables or provided by contacted authors).

Inter-rater agreement

To calculate inter-rater reliability, the first two authors double coded 17% of the articles (k = 148, of the k = 853 full articles assessed for eligibility). This resulted in a good inter-rater reliability, reflected in high intra-class correlations for continuous variables (ranging between 0.78 for age to 0.99 for sample size) and high Cohen's Kappa for categorical variables (ranging between 0.86 for informant, and 1.00 for country of the study). In case of disagreement, in-depth discussions were held to reach agreement on the specific content of the article. Full consensus was reached on all variables, providing us the confidence to divide the remaining articles between the two of us. The remaining 83% was divided equally among both authors.

Theoretical moderators

Age. We coded age at assessment continuously. For studies not reporting age but school grade, the average age of students in that school year was coded. For example, when the study mentioned adolescents were in sixth grade in the USA, we coded mean age as 11.5 years.

Adolescent gender. The proportion of boys and girls participating in the study was continuously coded, coding the percentage of girls in the sample (which could range from 0% to 100%). For example, studies with 60 girls and 40 boys was scored as 60%. Studies reporting effect sizes separately for boys and girls, adolescent gender was coded as 0% and 100%, respectively.

Country. The influence of country was assessed by applying the Hofstede dimensions, which are frequently applied in meta-analyses to examine the generalizability of an association across cultures (Parks-Leduc et al., 2015; Piotrowska et al., 2015). Countries were coded according to Hofstede's individualism score, power distance score, and femininity-masculinity score (Hofstede & McCrae, 2004 or see https://geert-hofstede.com/countries.html): (i) the individualism score reflects the extent to which identity is based on self-orientation and emphasis on individual achievement and initiative rather than collectivism (i.e., identity based on group orientation with emphasis on social system and belonging); (ii) power distance reflects a country's attitude towards the unequal distribution of power; (iii) masculinity reflects the extent to which a society is driven by competition, and achievement rather than by an emphasis on quality of life.

Methodological moderators

Time lag between family violence and self-control. For every study, we coded the time lag between the assessment of family violence and the assessment of self-control continuously in years (starting with a code of 0 for cross-sectional studies).

Informants. *For every effect size, we coded whether family violence was assessed by adolescents themselves (1 =* self-report*), by someone else such as one of the parents (2 =* other-report*), or whether the measure was a composite of different informants (3 =* composite*). Similarly, informant of the self-control measure was coded according to the reporting informant (1 =* self-report*, 2 =* other-report, such as parent report*, 3 =* composite of measures*, for example combination between self- and parent-report).*

Furthermore, studies were coded with 1 = consistent, when family violence and self-control were assessed by the same informant (e.g., both by adolescents themselves) and coded with 2 = inconsistent, when family violence

and self-control were assessed by different informants (e.g., family violence by parents and self-control by adolescents themselves). Important to note is that when both consisted of composite measures, specific attention was paid to check whether these composite scores comprised of the same informants. For example, a code of 1 was given when violence was measured with a composite score consisting of self-report and mother report and self-control was also measured with a composite score consisting of self-report and mother report. However, when violence was measured with a composite score of self-report and parent report and self-control with a composite score of self-report and teacher-report, a score of 2 was given.

Effect sizes

We obtained Pearson correlation coefficients to examine the strength of the association between family violence and adolescent self-control. The correlations were either derived from the studies or retrieved upon request if they were not present in the published paper. For consistency, we recoded effect sizes in which self-control was assessed as 'lack of self-control' or 'low self-control'. For normalization and standardization, correlations were transformed into Fisher's Z scores ES_z (Lipsey, & Wilson, 2001). The ES_z were the input for the analyses; after the analyses they were transformed back to *r* for interpretation (see Appendix A). Categorical moderator variables were dummy-coded with *k*—1 dummy variables (Assink & Wibbelink, 2016).

Publication bias

To take the possibility of publication bias into account, we created a funnel plot and performed an Egger's test on the effect sizes. The funnel plot allowed us to inspect the distribution of the effect sizes by displaying each individual effect size in a figure with the effect sizes on the horizontal axis and study precision as a function of standard errors on the vertical axis (Torgerson, 2006). Publication bias would occur if the funnel plot displayed an asymmetrical distribution. In order to formally test whether there was an asymmetrical distribution of effect sizes, we conducted an Egger's regression test (Egger, Davey Smith, Schneider, & Minder, 1997).

Data analyses

We performed all our analyses in R version 3.4.2 (R Core Team, 2017), using the Metafor package (Viechtbauer, 2010). Because most studies reported multiple effect sizes, there was a likely dependency between effect sizes derived from

the same studies (e.g., these effect sizes are not independent as they are part of the same sampling process, study group, and study population). To take this dependency into account, we applied a three-level meta-analysis, an approach that allows us to use all available information (i.e., multiple effect sizes, Assink & Wibbelink, 2016; Hendriks, Van der Giessen, Stams, & Overbeek, 2018; Van den Noortgate, López-López, Marín-Martínez, & Sánchez-Meca, 2013).

The three-level model specifies the following levels of variance: (1) sampling variance of the effect sizes, (2) variance between effect sizes within studies using the same dataset, and (3) variance between studies (Van den Noortgate et al., 2013). Studies using the same dataset are treated as if they all come from the same study. In this approach, studies with multiple effect sizes will not necessarily be assigned more weight because the dependency between effect sizes is taken into account. In contrast to the "classic" meta-analytic approach, selecting only one effect size from a single study or averaging effect sizes within studies, the three-level meta-analysis allows to include all effect sizes while taking the dependency into account. Doing so enables researchers to retain a maximum of information and achieve greater statistical power (Cheung, 2014; Hendriks et al., 2018; Van den Noortgate et al., 2013).To take into account possible dependency between studies using the same dataset, we used the number of independent studies (i.e., data sets) as the mode of analysis (Assink & Wibbelink, 2016).

To examine the association between family violence and adolescent self-control and moderator effects, we performed the following analyses. First, we estimated the overall mean effect size of the association. Second, we assessed between-study and within-study variance using a likelihood ratio test, and partitioned the total variance into percentages for the sampling variance, variance within studies, and variance between studies, applying earlier proposed methods (Cheung, 2014; Hendriks et al., 2018; Van den Noortgate et al., 2013). Third, based on whether there was evidence for heterogeneity among effect sizes, we performed univariate-moderator analyses. Fourth, we conducted multivariate moderator analyses to assessing significance of each moderator while considering other significant moderators to avoid multicollinearity problems in the analyses. The analyses were performed in line with earlier described procedures (Assink & Wibbelink, 2016), estimating parameters using restricted maximum likelihood.

RESULTS

Descriptives

The present meta-analysis included 28 studies reporting on the association between family violence and self-control. All included information is presented in an Excel table, facilitating opportunities for other scholars to use, update or extend our data for future research purposes (see online available data file, Supplementary Materials). Family violence included measures of severe punishment, slapping/hitting, physical coercion, severe verbal fights within the family, heatedly shouting and criticizing within the family, expressive anger and frequency of violence. It included general family violence, marital violence and parent-child violence. Self-control included measures of self-control, self-regulation, self-restraint, effortful control and persistence.

Of the 28 studies, 25 reported on independent studies, including 143 effect sizes and a total sample size of N = 26,214. Studies were published in a wide range of journals, for example in the Journal of Family Studies, Journal of Youth and Adolescence and Journal of Crime and Delinquency, and were published between 1990 and 2017. Most studies were conducted in the USA, followed by studies conducted in Asia and Europe. Age of the participating adolescents ranged between 10.00 and 21.70 years, with a mean age of 13.41 years (See Table 1 for more details). Most studies reported cross-sectional associations (26 studies, 104 effect sizes), with 5 studies (39 effect sizes) reporting longitudinal associations from family violence to self-control.

Studies focusing on the effect from self-control to family violence were scarce. Of the 28 included studies, we only identified three studies reporting longitudinal associations where self-control was measured first and family violence at a subsequent time point. While some argue three studies are enough to meta-analyze an effect, parameter estimates are poor when the number of studies is below five (Weisz et al., 2017). As a result, we could not meta-analyze the magnitude of the effect from self-control to family violence nor could we address the question regarding reverse causality, namely whether the magnitude of the directional effects differed. The results, therefore, only present cross-sectional effect sizes and longitudinal effect size from family violence to self-control.

Table 1. Descriptives

Variable	Characteristics	Descriptives
Studies included	*K* studies	28
	K independent	25
	N effect sizes	143
Publication year	Range	1990–2017
Journals	Range	20 different journals, e.g., Journal of Crime and Delinquency, Journal of Family Psychology, Journal of Youth and Adolescence
Dataset	Including	Flourishing families project, Healthy Families America (HFA) San Diego study, Longitudinal Study of Australian Children (LSAC), NICHD Study of Early Child Care and Youth Development (SECCYD), the Family and Community Health Study (FACHS)
Sample Size	Total sample	26,214
	Min sample size	65 (Feldman & Wentzel, 1990), 120 (Brody & Ge, 2001)
	Max sample size	3797 (Rowe, Zimmer-Gembeck, & Hood, 2016), 6429 (Beckmann, Bergmann, Fischer, & Mossle, 2017)
Age	Mean	13.41
	Min–Max	10–21.7
Adolescent gender	Overall balanced	87 effect sizes *(k = 20)*
	>60% boys	22 effect sizes *(k = 7)*
	>60% girls	34 effect sizes *(k = 6)*
Countries	Australia	1
	Hong Kong	2
	Germany	1
	Israel	1
	South Korea	1
	Switzerland	1
	UK	1
	USA	20
Hofstede individualism	Range	18 (South-Korea)–91 (USA)
Hofstede power distance	Range	13 (Israel)–68 (Hong Kong)
Hofstede masculinity	Range	39 (South-Korea)– 70 (Switzerland)
Time lag between family violence and self-control	Cross-sectional Longitudinal	104 effect sizes *(k = 26)* 39 effect sizes *(k = 5)*
	Average	1.30 years
Informant family violence	Self-report	79 effect sizes (*k* = 18)
	Other report	6 effect sizes *(k = 4)*
	Composite	54 effect sizes *(k = 6)*
Informant self-control	Self-report	56 effect sizes *(k = 18)*
	Other report	59 effect sizes *(k = 7)*
	Composite	20 effect sizes *(k = 3)*
Consistency	Consistent	67 effect sizes *(k = 17)*
	Inconsistent	76 effect sizes *(k = 13)*

Publication bias

As shown in Figure 2, the distribution of the effect sizes in the funnel plot appeared to be symmetrical. In addition, the Egger's test was non-significant Z = −0.994, p = 0.320. This suggested that there was no publication bias in the present meta-analysis.

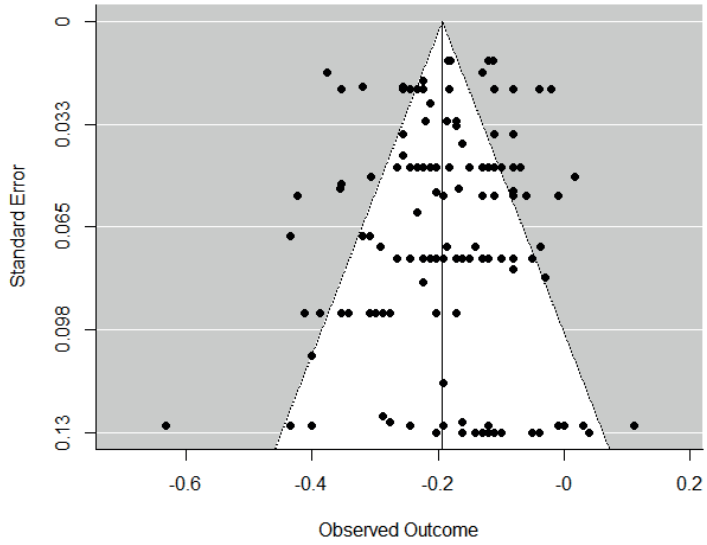

Figure 2. Funnel plot.

Overall effect size

We found a negative small to medium significant overall effect size for the association between family violence and adolescent self-control (ES_z = −0.194, S.E. = 0.015, t = −12.982, p < 0.001, 95% CI = (−0.223, −0.164), r = −0.191). This indicated that more family violence is significantly associated with lower adolescent self-control.

Moderator analyses

We performed univariate moderator analyses; Table 2 displays the statistics for the results. Significant moderators were age (QE(140) = 901.684, p < 0.001; Omnibus test: F(1, 140) = 8.913, p = 0.003) and time lag between family violence and self-control (QE(140) = 836.663, p < 0.001; Omnibus test: F(1, 140) = 8.367, p = 0.004). We explored the possibility that age as a moderator would show a non-linear pattern. Comparing age with a linear pattern versus

age with a non-linear pattern indicated the linear pattern to fit the data best (cf. lower Akaike Information Criterion (AIC) value for the linear pattern). The other moderators were not significant, including adolescent gender, Hofstede's scores, informant family violence, informant self-control, and consistency in informants.

Based on the significant moderators found in the previous analyses (age and time lag between family violence and self-control), we conducted a follow-up comparison as summarized in Table 3. Regarding age (centered at age 10), we found a significant effect (β_0 = −0.249, S.E. = 0.024, t = −10.288, p < 0.001, 95% CI = (−0.297, −0.202), r = −0.243), and a significant and positive slope (β_1 = 0.015, S.E. = 0.005, t = 2.985, p < 0.001, 95% CI = (0.005, 0.025)). This indicates a decrease in the magnitude of the association as adolescents get older (the constant is negative, and the positive slope will thus mitigate the starting value).

Regarding time lag between family violence and self-control, we found a significant effect (β_0 = −0.201, S.E. = 0.015, t = −13.505, p < 0.001, 95% CI = (−0.230, −0.171), r = −0.198) and a significant positive slope (β_1 = 0.036, S.E. = 0.012, t = 2.893, p = 0.004, 95% CI = (0.011, 0.061)). This indicates that the longer the time in-between measurements, the smaller the effect size.

Multiple moderator model

We conducted a multiple moderator model including both significant moderators from the univariate moderator analyses to assess their unique contribution (i.e., time lag between family violence and self-control, and age). The results of this multivariate model are summarized in Table 4. The significant omnibus test (F(2, 139) = 8.459, p < 0.001) suggested that at least one of the parameter estimates of the moderators significantly deviated from zero. Subsequent ANOVA tests indicated that both age and time lag had unique moderating effects on the relationship between family violence and self-control.

Table 2. Assessing moderators: The QE statistics illustrating residual heterogeneity, and the Omnibus to test the effect of the moderators on the family violence-self-control association.

Moderator	k_i	*N* ES	Omnibus Test	*p*	QE (df)	*p*
Age	24	142	F(1, 140) = 8.913 **	0.003	901.684 (140)	<0.001
Adolescent gender	25	143	F(1, 141) = 0.319	0.573	1011.77 (141)	<0.001
Hofs. Individualism	25	143	F(1, 141) = 0.195	0.659	1017.332 (141)	<0.001
Hofs. Power distance	25	143	F(1, 141) = 0.997	0.320	1009.720 (141)	<0.001
Hofs. Masculinity	25	143	F(1, 141) = 0.049	0.825	999.909 (141)	<0.001
Time lag	24	142	F(1, 140) = 8.367 **	0.004	836.663 (140)	<0.001
Informant fv	23	139	F(2, 136) = 0.377	0.687	898.725 (136)	<0.001
Informant sc	25	135	F(2, 132) = 0.326	0.326	923.373 (132)	<0.001
Consistency	25	143	F(1, 141) = 0.214	**0.644**	**1016.895 (141)**	**<0.001**

Note: **—p < 0.01, k_i —number of independent studies, N ES – number of effect sizes, Hofs—Hofstede's scores. Time lag—time lag between family violence and self-control, fv—family violence, sc—self-control.

Table 3. Univariate analyses presenting slopes of the significant moderators.

Moderators	*N* ES	ES_z	*SE*	*T*	95% CI	*p*	*r*
Age	142	−0.249	0.024	−10.288	(−0.297, −0.202)	<0.001	−0.244
		0.015	0.005	2.985	(0.005, 0.025)	0.003	
Time lag	142	−0.201	0.015	−13.505	(−0.230, −0.171)	<0.001	−0.201
		0.036	0.012	2.893	(0.011, 0.061)	0.004	

Note: Age was centered at age 10 (the minimum age), Time lag = time between family violence and self-control

Table 4. Results for the multiple moderator model

Moderator Variables	ES_z (SE)	95% CI	*t*-Statistic	*p*-Value
Intercept	−0.248 (0.022) **	(−0.291, −0.204)	−11.334	<0.001
Age	0.013 (0.005) **	(0.004, 0.022)	2.793	0.006
Time lag	0.033 (0.012) **	(0.009, 0.057)	2.725	0.007
Omnibus test:	$F(2, 139) = 8.459$ **			
Variance level 2	0.050 **			
Variance level 3	0.020 *			
N ES	142			

Note: * — p <.05, ** p<.01. Time lag — time lag between family violence and self-control

DISCUSSION

In the present meta-analysis, we synthesized research on the association between family violence and self-control across adolescence. We included 28 studies, conducted in eight countries, containing 143 effect sizes, with a total sample size of N = 26,214. The findings from the three-level meta-analysis revealed that family violence and self-control are significantly, small to moderately, negatively associated (r = −0.191). This indicates that family violence and low self-control coincide.

Moderators

Moderator analyses revealed that the association between family violence and low self-control did not differ significantly across country, adolescent gender, and informant. We did find a linear moderator effect for age; the magnitude of the association between family violence and self-control decreased over the course of adolescence. This finding suggests that adolescents gradually transform from parent-dependent to self-sustaining independent individuals (Laursen, Coy, & Collins, 1998; Sameroff, 2010; Tiberio et al., 2016). As a result, the influence of family factors such as family violence on adolescents may decrease, while the role of other contextual factors may increase. In the context of adolescence, this could indicate that the peer context becomes of increasing importance, perhaps buffering the negative effects experienced within the family (Levendosky, Huth-Bocks, & Semel, 2002).

We also found a moderator effect for the time between the measurement of family violence and self-control, with decreasing effect sizes for studies with a longer time lag between the assessment of family violence and subsequent self-control. This is in line with earlier methodological studies on the link between family factors and self-control, similarly indicating that the association is stronger when measured concurrently as compared to longitudinal assessments (Pallini et al., 2018). This is likely a result of more intervening processes taking place along the way, waning the direct effects of family violence on adolescent self-control.

It is important to note that we should be cautious in interpreting the direction of the effect. The association between family violence and self-control is likely to reflect a transactional process by which family violence and adolescent self-control mutually affect each other (Bandura, 1999; Harold et al., 2017; Sameroff, 2010). As such, family violence is likely to decrease self-control, which is in turn likely to evoke or exacerbate family violence

(Finkenauer et al., 2018; Finkenauer et al., 2015). The present meta-analysis revealed that most of the longitudinal studies included an effect from family to adolescent, without examining the reverse effect. While the results of the present meta-analysis provide an interesting starting point suggesting a link between family violence and self-control, future research on the links between family violence, self-control and psychosocial problems in a time sequential design are recommended (for example through random intercept cross-lagged panel models (Hamaker, Kuiper, & Grasman, 2015).

Implications

Adolescents exposed to family violence show heightened vulnerability to decrements in physical, mental, and social wellbeing. Although linkages between family violence and various problems are well-established, the specific processes underlying these associations are poorly understood. Recent theoretical work proposes self-control to play an important role in explaining these links (Finkenauer et al., 2018; Finkenauer et al., 2015). On the one hand low self-control may function as a possible mechanism because it is affected by family violence and contributes to maintaining violence. On the other hand, low self-control is reliably related to poorer physical, mental, and social health and wellbeing (Caspi et al., 2016; Moffitt et al., 2011). Supporting these theoretical suggestions, we found a significant association between family violence and self-control across adolescence, suggesting that self-control may play an important role in the link between family violence and adverse outcomes. As such, researchers and clinicians can expect low self-control in the presence of family violence, as opposed to treating low self-control and family violence as separate problems. For instance, family-based therapies targeting both family violence and self-control may well result in increased adolescent well-being and better family functioning, yet controlled trials are necessary to confirm this suggestion.

Limitations

First, we did not distinguish between inter-parental, parent-child, sibling-child, and parent-sibling violence, because most studies reported on family violence as a general construct without specifically specifying the family (sub) relationships involved in the conflict. Few studies provided in-depth details to distinguish between different relational levels at which the violence occurred. While both witnessing violence and experiencing violence are considered as detrimental for adolescents (Herrenkohl, Sousa, Tajima, Herrenkohl, & Moylan,

2008), further research is recommended to more specifically describe, measure, and compare different types of violence and their association with self-control in adolescence (Harold & Sellers, 2018).

Second, it is important to acknowledge that, when investigating interactions within families, not only environmental but also genetic factors play a role (Harold et al., 2017). This is evidenced by studies reporting on the intergenerational transmission and the heritability of family violence (Ehrensaft & Cohen, 2012; van der Aa, Boomsma, Rebollo-Mesa, Hudziak, & Bartels, 2010), and the intergenerational transmission and the heritability of self-control (Bridgett et al., 2015; Willems et al., 2018a). As a result, it may be that the observed association is partly explained by common genetic factors that simultaneously influence both family violence and self-control (Boomsma, Busjahn, & Peltonen, 2002; Harold et al., 2017). To paint a more complete picture of the association, future studies that integrate genetically sensitive designs investigating both environmental and genetic influences on the association between family violence, self-control and psychosocial problems and wellbeing would be particularly helpful.

CONCLUSIONS

Self-control – the capacity to regulate thoughts, emotions, and behavior – is a core component of healthy adolescent development. Results from the current meta- analysis indicate that family violence and adolescent self-control are negatively related, especially among younger adolescents. Because low self-control and family violence are reliably related to poorer health and wellbeing across the lifespan, these findings underscore the importance of considering both contextual and individual factors in treatment and policy addressing family violence. Although family violence is linked with adolescent self-control, and this link is not affected by a broad variety of moderators, we did find that the effects are stronger in studies with a shorter time delay. The meta-analysis also identified important gaps in our knowledge on the influence of genetic factors and reverse causality thereby providing promising inroads to enhance our understanding of the association between family violence and adolescent self-control.

APPENDIX A

The Fisher's transformation of *r* was done using the following formula: $ES_Z = \frac{1}{2}\log_e\left[\frac{1+r}{1-r}\right]$. Any ES_Z can be transformed back into standard correlation form using the inverse of the ES_Z transformation using the following formula: $r = \frac{e^{2ESZ}-1}{e^{2ESZ}+1}$ [63].

Chapter 4

The heritability of self-control: A meta-analysis

ABSTRACT

Self-control is the ability to control one's impulses when faced with challenges or temptations, and is robustly associated with physiological and psychological well-being. Twin studies show that self-control is heritable, but estimates range between 0% and 90%, making it difficult to draw firm conclusions. The aim of this study was to perform a meta-analysis to provide a quantitative overview of the heritability of self-control. A systematic search resulted in 31 included studies, 17 reporting on individual samples, based on a sample size of >30,000 twins, published between 1997 and 2018. Our results revealed an overall monozygotic twin correlation of .58, and an overall dizygotic twin correlation of .28, resulting in a heritability estimate of 60%. The heritability of self-control did not vary across gender or age. The heritability did differ across informants, with stronger heritability estimates based on parent report versus self-report or observations. This finding provides evidence that when aiming to understand individual differences in self-control, one should take genetic factors into account. Recommendations for future research are discussed.

Keywords: Self-control, Twin, Heritability, Meta-analysis, Genetics

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One factor that contributes to good adjustment across the lifespan is self-control. Yet, not all individuals develop the same levels of self-control, which begs the question: 'where do these individual differences come from?'. While the effects of the environment on such individual differences are well documented (Bridgett, Burt, Edwards, & Deater-Deckard, 2015; Pallini et al., 2018; Willems et al., 2018b), the research on genetic influences on self-control is more nascent. The aim of this study is therefore to perform a meta-analysis to provide a quantitative overview of the heritability of self-control.

Self-control is defined as the capacity to alter unwanted impulses and behaviors in order to bring them into agreement with internal and external standards (Duckworth & Steinberg, 2015; Tangney, Baumeister, & Boone, 2004). Multiple studies across disciplines emphasize the importance of self-control. On the one hand, individuals with high self-control are happier, healthier, and wealthier across adolescence and adulthood, compared to those with low self-control (de Ridder, Lensvelt-Mulders, Finkenauer, Stok, & Baumeister, 2012; Duckworth & Seligman, 2005; Finkenauer, Engels, & Baumeister, 2005; Hofmann, Luhmann, Fisher, Vohs, & Baumeister, 2014). On the other hand, low self-control has been associated with lack of success in school, relationships, and the labor market (Caspi et al., 2016; Moffitt et al., 2011; Vazsonyi, Mikuška, & Kelley, 2017). Because self-control is a powerful predictor of health, wealth, and public safety, numerous studies examined why some individuals have higher self-control than others . Most of these studies focused on environmental effects, examining how parenting or peer involvement explains variation in self-control (Finkenauer, Engels, & Baumeister, 2010; Gottfredson & Hirschi, 1990; Karreman, Van Tuijl, van Aken & Deković, 2006; King, McLaughlin, Silk, & Monahan, 2018; Pallini et al., 2018).

In the last decade, though, various studies have shown that almost all traits and behaviors are at least partly influenced by genetic factors (Polderman et al., 2015). For self-control, results are mixed. For example, some studies state that differences in self-control are not or weakly explained by genetic factors (e.g., Friedman et al., 2011), while others state that almost all variation in self-control is explained by genetic factors (e.g., Beaver et al., 2009; Wright, Beaver, Delisi, & Vaughn, 2008), and again others state that about half of the variance in self-control is explained by genetic factors (e.g., Boisvert, Wright, Knopik, & Vaske, 2013; Willems et al., 2018a; Yamagata et al., 2005). To obtain a clearer picture from previously researched data of the genetic influence on self-control, we performed a meta-analysis including twin studies

that address the heritability of self-control. By doing so, we aim to provide an encompassing and quantitative overview on the extent to which genetic factors play a role in explaining individual differences in self-control.

METHODS

Twin design

The classical twin design is built on the premise that differences in the resemblance between monozygotic twins (sharing approximately 100% of their DNA) and dizygotic twins (sharing 50% of their segregating genes on average) can be used to parse phenotypic trait variance into genetic and environmental components. Genetic influences are implied if the correlation between monozygotic twin (MZ) pairs is higher than the correlation between dizygotic twin (DZ) pairs. An influence of the common environment – influences that are shared between family member– is implied when the DZ twin pair correlation is higher than half of the correlation between MZ twin pairs. Unique environmental factors are person specific and not shared between twins. Identical twin correlation's deviation from 1 provide a direct estimate for the non-shared environmental influences, since identical twins share both their genetic make-up as well as part of the environment (the shared environment).

More specifically, twin correlations can potentially be parsed into additive genetic (A), non-additive or dominance genetic (D), common environment (C), and non-shared environment (E). If MZ correlations are larger than DZ correlations, A, C, and E effects are to be expected. If MZ correlations are more than twice the DZ correlations non-additive genetic effects are expected. In the classical twin design the non-additive genetic influences (D) and shared environmental influences (C) are confounded and cannot be estimated in the same model, and it is common for authors to estimate one or the other based on the twin correlations. Note that this distinction does not influence the results presented in our study as we speak of general genetic influence without specifically modelling the difference between additive or non-additive genetic influences.

Like any statistical model, the classical twin design is based on certain assumptions. One key assumption of twin models is that of the "equal environment", assuming that the environment of monozygotic twins is no more similar than the environment of dizygotic twins. Critics of twin models state that the equal environment assumption does not hold, because MZ twins

receive more similar treatment, and that heritability estimates are therefore not trustworthy (e.g., Burt & Simons, 2014). However, empirical evidence (systematic reviews, simulation studies and twin studies, for example with twin with misclassified zygosity) shows that this assumption is typically not violated, with heritability estimates garnered in twin models being relatively unbiased (Barnes et al., 2014; Conley et al., 2013).

Search of studies

Articles were retrieved from various online databases through a computerized literature search. The databases included *PubMed* (http://www. ncbi.nlm.nih.gov/pubmed), *PsycINFO* (http://www.apa.org/pubs/databases/psycinfo/index.aspx) and *Web of Science* (http://apps.webofknowledge.com). A literature search was conducted for studies published up to March 28th 2018. The following search terms (and their variations) were used: 1) *twin* OR *heritability* OR *genetics*, as well as 2) *self-control* OR *self-regulation* OR *effortful control* OR *self-discipline*.

Selection procedure

Studies were eligible for this meta-analysis when the following criteria were met.

First, the study had to include twin correlations or standardized heritability estimates. This information is necessary to be able to extract information on the extent to which individual differences in self-control are explained by genetic factors. Second, the study had to assess self-control or a concept closely related to self-control, such as self-regulation, effortful control, self-discipline, or emotion regulation (Nigg, 2017). Third, only papers originally published in English and that were published in peer-reviewed journals were included. Fourth, we excluded papers that assessed individuals with clinical psychological problems (e.g. schizophrenia and autism), as well as papers primarily focusing on clinical physiological disorders (e.g. obesity and diabetes).

The initial search in the databases yielded a total of 6,375 unique hits. Titles and abstracts of these hits were examined according to the inclusion criteria, resulting in 160 papers that were selected for in-depth reading. We also inspected possible missing publications by the main authors of the identified papers, resulting in the identification of 11 additional publications. Subsequently, all 171 articles were screened according to the inclusion criteria, resulting in 31 articles to be included in the present meta-analysis (see Figure 1).

The main reasons for exclusion were that studies mentioned the heritability of self-control but did not contain a twin sample (60%), or did not empirically assess self-control (30%). Additionally, a substantial number of the excluded articles did not provide MZ/DZ correlations or other heritability measures needed to infer the genetic and environmental effects on self-control (10%). References included in this systematic review are preceded by * in the reference list.

Coding the studies

The first two authors coded all 31 articles, retrieving descriptive information (authors, article title, journal, year of publication), sample information (country, cohort, sample size, age), methodological information (measurement of self-control, informant of the measure), and heritability estimates (MZ correlation, DZ correlation, and standardized heritability estimate of the overall model if provided and otherwise estimates of the best fitting model). For every twin correlation, we coded age (1=*early childhood, 0-6 years;* 2= *middle childhood, 7 - 12 years;* 3= *adolescence, 13-18 years;* 4= *adulthood, 18+ years*), and informant (1= *parent report,* 2= *self-report,* 3=*observation*). Some studies provided twin correlations separately for boys and girls. These studies were coded accordingly (1=*girls* 2=*boys*). See Table 1 for an overview and description of all the included papers.

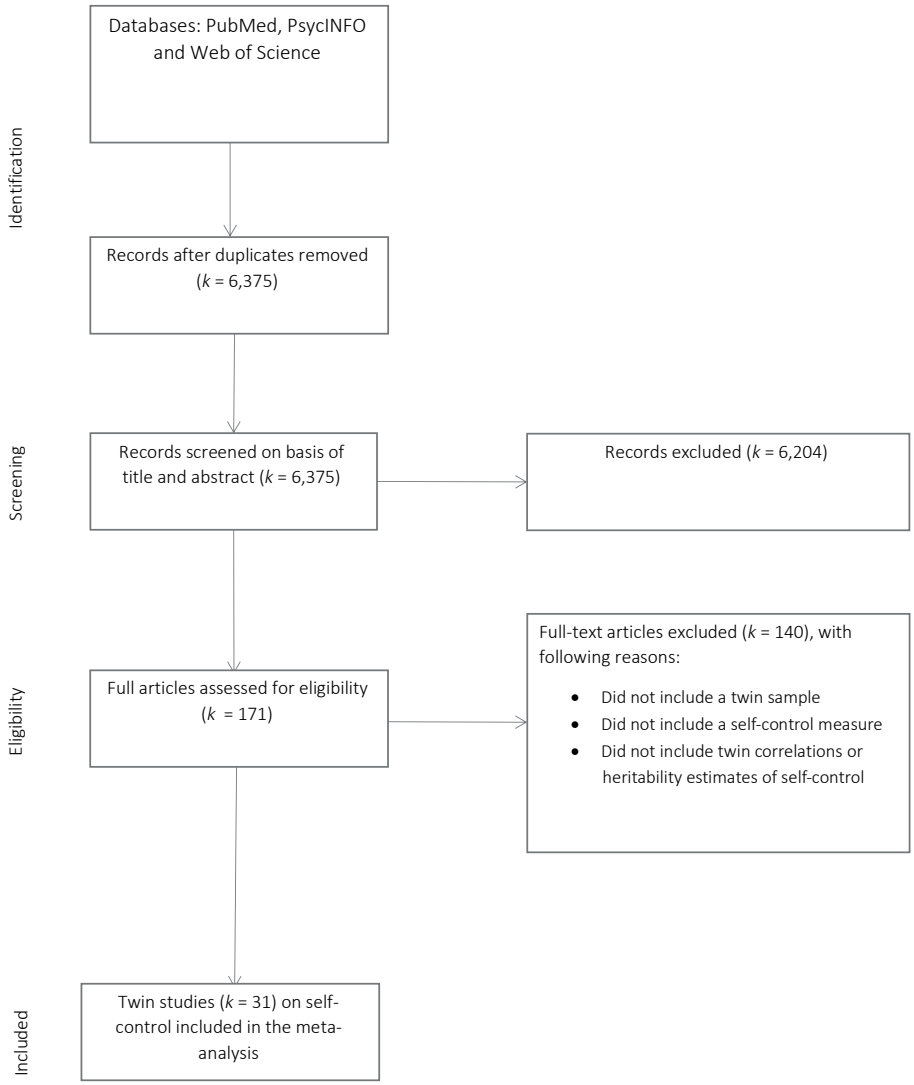

Figure. 1. PRISMA flowchart of selected twin studies.

Table 1. Overview of the included heritability studies.

Author(s)	Country	Cohort	SC term	Measure	Age	Informant	Sex	MZ	DZ	rMZ	rDZ	A (95% CI)	C (95% CI)	E (95% CI)	D (95% CI)
Anohkin *et al.* (2011)	USA	LTGBFB	DD	DoG	12	Lab task	M	82	71	.45	.02	.30 (.15-.49)		.70 (.51-.85)	
							F	87	49	.38	.29				
							OS		83		.34				
					14	Lab task	M	63	50	.82	.14	.51(.28-.71)		.49 (.29-.72)	
							F	66	31	.46	-.14				
							OS		56		.17				
Beaver (2008)	USA	AH	SC	SoI	15	Self	All	245		.41					
					16	Self		190		.32					
Beaver *et al.* (2008a.)	USA	ECLS-K	SC	SoIssrs	4	Par/Teach	All	55	95	.72	.36	.72 (.58-.86)		.28 (.14-.42)	
					6	Par/Teach				.46	.23	.46 (.20-.73)		.54 (.27-.80)	
Beaver *et al.* (2008b.)*	USA	AH	SC	SoI	15	Self	All	289	452	.64	.32	.64	0	.36	
					16	Self				.53	.27	.53	0	.47	
Beaver *et al.* (2009)	USA	AH	SC	SoI	15	Self	All	300	400	.41	.12				
					16	Self				.32	.12				
Beaver *et al.* (2013)	USA	CNLSY	SC	BPI	9	Par	All	13	1664	.94	.49	.90 (.79-1.00)	.04 (-.05-.13)	.06 (.02-.08)	
					11	Par				.82	.56	.53 (.35-.71)	.29 (.18-.40)	.18 (.04-.26)	
					13	Par				.88	.53	.71 (.47-.94)	.16 (.00-.32)	.12 (.00-.23)	
Boisvert *et al.* (2012)	USA	AH	SC	SoI	15	Self	All	307	477	.45	.24	.42 (.19-.52)	.02 (.00-.20)	.55 (.48-.65)	
					16	Self				.35	.14	.43 (.19-.52)	.02 (.00-.20)	.55 (.47-.65)	
					18.5	Self				.50	.11	.43 (.18-.52)	.02 (.00-.21)	.56 (.48-.65)	
Boisvert *et al.* (2013a)	USA	AH	SC	SoI	15	Self	All	289	452	.39	.23	.30 (.04 - .45)	.08 (.00- .27)	.62 (.52- .73)	
					16	Self				.44	.16	.40 (.20-.49)	.00 (.00-.15)	.60 (.51 - .70)	
					22	Self				.42	.12	.35 (.15 - .46)	.00 (.00-.15)	.64 (.54-.75)	
					29	Self				.39	.20	.37 (.07 -.47)	.01 (.00-.23)	.62 (.53- .74)	

Author(s)	Country	Cohort	SC term	Measure	Age	Informant	Sex	MZ	DZ	rMZ	rDZ	A (95% CI)	C (95% CI)	E (95% CI)	D (95% CI)
Boisvert *et al.* (2013b)	USA	AH	SC	SoI	15	Self	M	155	124	.33	.29	.09 (.00-.46)	.22 (.00-.40)	.69 (.54-.82)	
							F	149	118	.49	.30	.44 (.01-.63)	.08 (.00-.42)	.48 (.36-.64)	
					16	Self	M			.53	.23	.47 (.01-.59)	.00 (.00-.40)	.52 (.41-.67)	
							F			.50	.15	.47 (.13-.59)	.00 (.00-.27)	.53 (.41-.68)	
					22	Self	M			.43	.18	.61 (.48-.78)	.00 (.00-.39)	.59 (.45-.76)	
							F			.42	.17	.39 (.00-.52)	.00 (.00-.39)	.61 (.48-.78)	
Boutwell *et al.* (2013)	USA	AH	SC	SoI		Self	All	289	450	.38	.22	.38 (.13-.49)	.02 (.00-.18)	.60 (.51-.71)	
Boutwell *et al.* (2014)	USA	AH	SC	SoI	22	Self	All	285	247	.40	.17	.37 (.04-.48)	.01 (.00-.28)	.62 (.52-.74)	
							M	144	131	.41	.13	.38 (.00-.52)	.00 (.00-.32)	.62 (.48-.79)	
							F	145	116	.35	.18	.27 (.00-.47)	.06 (.00-.37)	.66 (.52-.83)	
Coyne *et al.* (2014)	USA	ECLS-K	SC	SoIssrs	5.72	Par/Teach	All	59	121	.73	.56	.35 (.08-.62)	.38 (.14-.61)	.27 (.18-.36)	
					7.26	Par/Teach				.64	.42	.44 (.01-.87)	.20 (-.16-.56)	.36 (.23-.49)	
					9.27	Par/Teach				.56	.32	.49 (.01-.97)	.07 (-.31-.45)	.44 (.24-.63)	
					11.26	Par/Teach				.67	.34	.67 (.53-.81)		.33 (.19-.48)	
Deater-Deckard *et al.* (2007)	USA	WRRP	EC	BBR	6.09	Obs	All	105	154	.51	.30	.28 (-.06-.56)	.22 (.00-.40)	.50 (.39-.62)	
Fagnani *et al.* (2017)	IT	ITS	SC	SoIA	16.3	Par/Self	All	281	493	.59	.29	.58 (.47-.67)		.42 (.33-.53)	
Friedman *et al.* (2011) *	USA	CLTS	SRS	Stroop;stop signal	1.2	Obs	All	435	378	.50	.33	.36	.15	.49	
					1.7	Obs				.36	.33	.06	.29	.65	
					2	Obs				.23	.29	.00	.26	.74	
					3	Obs				.53	.33	.40	.13	.47	
Gagne *et al.* (2010)	USA	BUTP	IC	Lab-TAB	2.07	Obs	All	133	161	.38	.16	.38 (.06-.51)	.00 (.00-.23)	.62 (.49-.77)	
						Par				.84	.55	.58 (.39-.82)	.26 (.03-.44)	.16 (.12-.21)	

Author(s)	Country	Cohort	SC term	Measure	Age	Informant	Sex	MZ	DZ	rMZ	rDZ	A (95% CI)	C(95% CI)	E (95% CI)	D (95% CI)
Gagne *et al.* (2011a.)	USA	WSBR	IC	Lab-TAB	3	Obs	All	173	338	.49	.33		.37 (.28-.49)	.63 (.51-.72)	
				CBQ		Par		130	237	.67	.21				
Gagne *et al.* (2011b.)	USA	BUTP	IC	Lab-TAB	2.07	Obs	All	131	160	.41	.15	.39 (.25-.51)		.61 (.49-.75)	
				TBAQ-R		Par				.85	.55	.67 (.51-.85)	.16 (.01-.31)	.17 (.13-.22)	
Gagne *et al.* (2016)	USA	BUTP	IC	TBAQ-R	2	Par	All	145	168	.87	.55	.61 (.78-.87)	.23 (.01-.41)	.17 (.13-.22)	
				Lab-TAB		Obs				.38	.12	.38 (.14-.51)	.00 (.00-.18)	.62 (.49-.76)	
				TBAQ-R	3	Par	All			.73	.42	.23 (.08-.45)	.13 (.00-.33)	.01 (.00-.04)	
				Lab-TAB		Obs				.26	.35	.06 (.00-.25)	.00 (-.36 -.36)	.01 (-.04 -.00)	
Gagne *et al.* (2017)	USA	WTP	AC	EATQ; HBQ	13.6	Par/Self	All	188	258	.59	.15	.73		.27	
							M			.57	.17				
							F			.61	.13				
Goldsmith *et al.* (1997)	USA		PER	TBAQ	2.21	Par	All	89	95	.81	.33				
				CBQ	5.54	Par		55	64	.53	.24				
Guo *et al.* (2011)	CN		EC	EATQ-R	13.74	Self	All	585		.44					
						Par				.71					
Gustavson *et al.* (2014)	USA	CLTS	IM	UPPS-P		Self	All	181	166	.48	-.03				
			SC	UPPS-P Self						.47	.13				
			PR	GPS Self						.56	.07				
			VO	VCI Self						.39	.10				
			ACT	ACS Self						.32	.05				
Hahn *et al.* (2016)	DE	GTSIO	SD	TCI	33.3	Self	All	146	90	.59	.16	.54 (.00-.15)	.00 (.00-.15)	.41 (.32-.53)	
			LoC	GPIUS	33.3	Self				.37	.26	.21 (.00-.50)	.16 (.00-.43)	.63 (.50-.77)	
			MR	GPIUS	33.3	Self				.41	.28	.33 (.00-.54)	.09 (.00-.42)	.58 (.46- .73)	
			SR	GPIUS	33.3	Self				.26	.18	.21(.00- .41)	.06 (.00-.33)	.73 (.59-.88)	
Lemery-Chalfant *et al.* (2008) *	USA	WTP	EC	CBQ	7.58	Par	M	214	198	.71	- .11	.00		.32	.68

Author(s)	Country	Cohort	SC term	Measure	Age	Informant	Sex	MZ	DZ	rMZ	rDZ	A (95% CI)	C (95% CI)	E (95% CI)	D (95% CI)
							F			.65	.27	.00		.32	.68
							OS		151		.04				
				CBQ	7.88	Par	M			.67	.03	.41		.21	.38
							F			.75	.32				
							OS				.04				
				BRS		Obs	M			.82	.44	.83		.17	.00
							F			.82	.50				
							OS				.52				
Li *et al.* (2014)	CN	BTS	EC	EATQ-R	15.47	Self	M	279	97	.58	.38				
						Par				.30	.45				
						Self	F	328	121	.59	.22				
						Par				.43	.25				
Wang *et al.* (2013)	USA	BUTP	ER	BSID II	2.99	Obs		140	164	.53	.27	.43 (.16-.58)	.09 (.00-.30)	.48 (.39-.60)	
Willems *et al.* (2018a)	NL	NTR	SC	ASCS	7	Mot	M	2050	2075	.74	.34	.59 (.21- .77)		.25 (.24- .27)	.16 (-.02- .34)
							F	2286	1906	.70	.32	.51 (.36- .67)		.31 (.29- .33)	.18 (.02- .34)
							OS		3871		.31				
						Fa	M	1453	1482	.75	.39	.75 (.68- .82)		.25 (.23- .27)	.01 (-.06- .07)
							F	1671	1300	.73	.36	.68 (.54- .82)		.27 (.26- .29)	.04 (-.09- .18)
							OS		2684		.32				
						Teach	M	881	887	.61	.32	.56 (.37- .75)		.36 (.32- .39)	.08 (-.11- .28)
							F	992	802	.63	.17	.29 (.14- .44)		.37 (.35- .39)	.34 (.19- .49)
							OS		1631		.27				
					10	Mot	M	1636	1572	.73	.36	.69 (.49- .88)		.25 (.24- .27)	.06 (-.14- .26)
							F	1867	1463	.71	.32	.48 (.37- .59)		.28 (.26- .30)	.25 (.14 - .36)
							OS		3182		.32				
						Fa	M	1150	1083	.76	.35	.67 (.52- .82)		.24 (.21- .26)	.10 (-.05 -.24)

Author(s)	Country	Cohort	SC term	Measure	Age	Informant	Sex	MZ	DZ	rMZ	rDZ	A (95% CI)	C(95% CI)	E (95% CI)	D (95% CI)
							F	1299	987	.70	.40	.70 (.63- .77)		.29 (.27- .31)	.01 (-.06 -.08)
							OS		2161		.31				
						Teach	M	813	770	.66	.33	.65 (.55- .75)		.32 (.16- .48)	.03 (-.23 -.29)
							F	912	705	.66	.27	.35 (.10- .59)		.31 (.27- .34)	.35 (.07 - .63)
							OS		1559		.22				
					12	Mot	M	1411	1337	.75	.34	.57 (.40- .74)		.26 (.24-.28)	.17 (.00 - .34)
							F	1600	1274	.73	.37	.74 (.66 - .81)		.26 (.24 - .27)	.01 (-.06 .08)
							OS		2676		.32				
						Fa	M	988	938	.78	.41	.78 (.76 - .80)		.22 (.20 - .24)	.00 (.00 - .00)
							F	1142	899	.73	.40	.73 (.71 - .74)		.27 (.26 -.29)	.00 (.00 - .00)
							OS		1859		.35				
						Teach	M	633	608	.67	.35	.69 (.65 - .73)		.31 (.27 - .35)	.00 (-.02 -.02)
							F	798	560	.63	.31	.39 (.09 - .70)		.34 (.31 - .36)	.27 (-.03 -.58)
							OS		1135		.27				
						Self	M	172	157	.57	.32	.39 (.23 - .54)		.43 (-.05 -.91)	.18 (-.18 -.54)
							F	197	144	.40	.32	.26 (.04 - .49)		.58 (.03-1.00)	.16 (-.18 -.49)
							OS		182		.03				
					14	Self	M	739	670	.44	.19	.38 (.18 - .58)		.57 (.55 - .60)	.05 (.15 - .25)
							F	1103	837	.52	.21	.32 (.06 - .58)		.47 (.43 - .52)	.21 (-.06 -.47)
							OS		1661		.16				
					16	Self	M	565	461	.45	.23	.40 (.04 - .76)		.50 (.47 - .53)	.10 (-.26 -.46)
							F	868	666	.44	.15	.20 (.00 - .41)		.52 (.47 - .58)	.28 (.05 - .50)

Author(s)	Country	Cohort	SC term	Measure	Age	Informant	Sex	MZ	DZ	rMZ	rDZ	A (95% CI)	C (95% CI)	E (95% CI)	D (95% CI)
							OS		1223		.20				
Wright *et al.* (2008) *	USA	AH	SC	SoI	15	Self	All	289	452	.44	.34	.40	.00	.60	
					16	Self				.18	.07	.25	.01	.74	
Yamagata *et al.* (2005) *	JP	KTP	EC	EC scale	24.15	Self	All	152	73	.45	.21	.49		.51	
			ACC	EC scale		Self				.38	.17	.39		.61	
			ATC	EC scale		Self				.42	.20	.45		.55	
			IC	EC scale		Self				.30	.12	.32		.68	
Yancey *et al.* (2013)	USA	UMTR	TI	ESI	29.4		All	130	124	.60	.24	.59 (.46-.69)	.00	.41 (.31-.54)	

Abbreviations: ***Countries:*** CN – China, DE – Germany, IT –Italy, JP – Japan, NL – the Netherlands, USA – United States of America; ***Cohorts:*** AH – Add Health, BPI –the Behavioral Problems Index, BUTP – Boston University Twin Project, BTS – Beijing Twins Study , CNLSY – the Child and Young Adult Supplement of the National Longitudinal Survey of Youth 1979, CLTS – Colorado Longitudinal Twin Study, ECLS-K – the Early Childhood Longitudinal Survey, Kindergarten Class, GTSIO – German Twin Study on Internet and Online-game behavior, ITS – Italia Twin Register, KTP – Keio Twin Project, LTGBFB – Longitudinal Twin Study of the Genetics of Brain function and Behavior, NTR – the Netherlands Twin Register, WRRP – Western Reserve Reading Project , WSBR – Wisconsin State Birth Records, WTP – Wisconsin Twin Project, UMTR – University of Minnesota Twin Registry; ***Self-control Term:*** AC – Attentional Control, ACT – Action Control, DD – Delay Discounting, EC – Effortful-Control, ER – Emotion Regulation, IC – Inhibitory Control, IM – Impulsivity, LC – Locus of control, PR– procrastination, PER – Persistence, SC – Self-Control; SR – Self-Regulation, SRS – Self-Restraint, SD – Self-Directedness, TCI – Temperament and Character Inventory, Temp – Temperament, VO – Volitional behavior; ***Measure:*** ACS – Action Control Scale, ASCS – ASEBA Self-Control Scale, BBR – Bayley's Behavior Records, BRS – Bayley Rating Scale, CBQ –Child Behavior Questionnaire , BSID-II – the Bayley Scales of Infant Development-Second Edition, DoG – Delay of Gratification procedure, EATQ(-R) – Early Adolescent Temperament Questionnaire (Revised), EC Scale – Effortful Control Scale, ESI – Externalizing Spectrum Inventory, GPS– General Procrastination Scale, GPIUS – Generalized Problematic Internet Use Scale , HBQ – the MacArthur Health and Behavior Questionnaire Lab-TAB – Laboratory Temperament Assessment Battery, SoI – Sum of Items (Add Health), SoIA – Sum of Items Achenbach scales, SoIssrs – Sum of Items Social Skills Rating Scale, TBAQ(-R) – the Toddler Behavior Assessment Questionnaire (Revisited), VCI – Volitional Components Inventory; ***Informant:*** fa – father report , mot—mother report, par – parent-report , self –self-report, teach – teacher report; ***Sex*****:** All – Males and Females, F – Females, M – Males, OS – Opposite sex. ***Heritability Parameters:*** MZ – monozygotic , DZ – Dizygotic, A – additive genetic, C – common environment, E – unique environment, D – dominant genetic influences. ***Note.*** Articles marked with an * did not report confidence intervals in article.

Analyses

The meta-analysis was performed in the Metafor package in R version 3.5.1 (Viechtbauer, 2010; R Core Team, 2017). Our dataset and statistical scripts can be accessed from the supplements, providing opportunities for other scholars to use, update or extend our data for future research purposes. Many studies reported multiple effect sizes, for example reporting twin correlations for different self-control measures, for different age groups, separately for boys and girls, and separately for parent-report and self-report. Additionally, multiple studies used data from the same cohort, for example multiple studies used the AddHealth data (http://www.cpc.unc.edu/projects/addhealth). As a result, it is likely that effect sizes from the same sample are more similar than effect sizes from different studies, as they are part of the same sampling process, study group, and study population. Previous meta-analyses only included one effect size of each included study to deal with this dependency (Bartels, 2015; de Zeeuw, de Geus, & Boomsma, 2015). More recently, multi-level meta-analyses are suggested to be more preferable, because they take the dependency between studies into account while including all effect sizes. Thereby it increases statistical power and provides maximum information of your data (Assink & Wibbelink, 2016; Hendriks, Van der Giessen, Stams, & Overbeek, 2018; Van den Noortgate, López-López, Marín-Martínez, & Sánchez-Meca, 2013). Accordingly, we applied a multi-level meta-analysis for the present study taking into account sampling variance (level 1), variance between effect sizes from the same sample (level 2), and variance between studies (level 3) (Hox et al., 2010; Van den Noortgate et al., 2013).

The analyses were conducted in multiple stages. First, we decided which effect size to meta-analyze. The included studies provide different parameters that allow to estimate the heritability of self-control, namely (1) MZ and DZ correlations or, (2) standardized genetic variance (cf. the heritability). Multiple studies only presented their best fitting model (dropping non-significant parameters), and report only the variance decomposition based on this best fitting model. This model choice and preference is sensitive to sample size, thereby possibly presenting a biased perspective (often an overestimation) of genetic influences on self-control (Posthuma & Boomsma, 2000). For the present study, we therefore decided to meta-analyze the twin correlations (MZ correlation and DZ correlation, respectively), rather than the standardized genetic variance.

Second, both the MZ correlations (r_{mz}) and the DZ correlations (r_{dz}) were transformed into Fisher's Z scores ES_Z (ES_{Zmz} and $ES_{Zdz,}$ respectively). This ES_Z

scores is assumed to approach normality, which is necessary for the accurate determination of mean effect size estimates and for unbiased tests of statistical significance (Lipsey & Wilson, 2001). Subsequently, we meta-analyzed the ES_{Zmz} and the ES_{Zdz} separately, resulting in an overall ES_{Zmz} and an overall ES_{Zdz}. Dependency between effect-sizes was taken into account by categorizing all effect sizes based on the same sample within the same level, in line with the multi-level meta-analytic approach (Assink & Wibbelink, 2016; Viechtbauer, 2010). So effect sizes based on the same sample received the same 'identification number' , to take into account the dependency between these effect sizes, which was used in the multilevel approach. Next, the Fisher's Z scores, ES_{Zmz} and $ES_{Zdz,}$ were transformed back to MZ correlations (r_{mz}) and DZ correlations (r_{dz}), for interpretation purposes (Lipsey& Wilson, 2001)[8]. Additionally, we calculated the heritability of self-control by applying Falconer's formula: $h^2=2(r_{mz}- r_{dz})$, with r_{mz} being the meta-analytic correlation of self-control among MZ twins and r_{dz} the meta-analytic correlation of self-control among DZ twins (Falconer, 1960). Third, we examined whether the ES_{Zmz} and ES_{Zdz} were potentially moderated by a number of factors such as gender, age, and informant.

RESULTS

Descriptives

A total of 31 papers were included (see Table 1 for an overview). Of the 31 papers, 17 papers reported on independent samples. Multiple articles applied data from the Add Health project (k=9), the Boston University Twin Project (k=4), and the Colorado Longitudinal Twin Project (k=2). Most studies were conducted in the United States of America (k=25 twin studies). The other studies (k=6) were based on non-American samples, with two studies from China (a population sample and the Beijing Twin study), one study from Germany (German Twin Study on Internet and Online Game Behavior), one study from Italy (Italia Twin Register), one study from Japan (Keio Twin Project), and one study from the Netherlands (the Netherlands Twin Registry), respectively. The total sample size, only counting sample size of independent studies, was 15,892 MZ individuals and 17,384 DZ individuals, with a total sample size of 33,276.

8 The Fisher's transformation of r was calculated in Excel (FisherZ function) using the following formula: $ES_{Z_r} = \frac{1}{2}\log_e\left[\frac{1+r}{1-r}\right]$. For the back transformation, the function FisherInv was applied using the following formula: $r = \frac{e^{2ESZr}-1}{e^{2ESZr}+1}$ (see Field, 2001; Lipsey & Wilson, 2001).

The earliest published paper was in 1997 (Goldsmith et al., 1997), while the most recent publication was in 2018 (Willems et al., 2018a). The papers were published in 20 different journals. Most studies used self-reports (*ES*= 46), or parent reports (*ES*=29), and some studies included observations (*ES*=17). In total, 20 different measures were used, such as the Children's Behavior Questionnaire (Ahadi, Hershey, & Fisher, 2001), and the ASEBA Self-Control scale (Willems et al., 2018a).

The smallest sample consisted of 119 twin pairs (Goldsmith et al., 1997), while the largest sample consisted of more than 4000 twin pairs (Willems et al., 2018a). The samples covered a wide age-range, from 1.20 years (Friedman et al., 2011) to 33.30 years (Hahn et al., 2016), with an average age of 13.04 years. Most studies reported on children in middle childhood (7-12 years, *ES*= 33) or adolescence (13-18 years, *ES*=33), but there were also studies specifically investigating early childhood (0-6 years, *ES*=21) and adulthood (>19 years, *ES*=20).

For the present study, we meta-analyzed the twin correlations (MZ correlation and DZ correlation, respectively), rather than the standardized genetic variance. Of the 31 included studies, 11 studies reported twin correlations of full models with correlations separately for males and females, 14 studies reported correlations for full models constraining correlations to be equal for males and females, 4 studies reported correlations for the best fitting model, and for 2 studies it was unclear whether the correlations were based on full or best fitting models.

Heritability of self-control

The 31 twin studies provided 108 MZ twin correlations and 104 DZ correlations (two studies only included MZ twins; Beaver et al., 2008; Guo et al., 2011). The MZ twin correlations ranged between .18 (Wright et al., 2008) and .94 (Beaver et al., 2013). The DZ correlations ranged between -.14 (Anohkin et al., 2010) and .56 (Coyne et al., 2014). An examination of the standardized heritability estimates showed that heritability ranged from 0% (Friedman et al., 2011, Gagne et al., 2011) to 90% (Beaver et al., 2013).

This heterogeneity in the heritability estimates is likely a result of the sample size of the studies. A scatterplot of the 31 studies, including the distribution of MZ and DZ correlations across sample sizes respectively, showed that there was less variance between studies with increasing sample size (see Figure 2). Studies with a small sample size showed more variance in the MZ correlations (with correlations ranging between .28 to .94) than studies with larger sample

sizes (with correlations ranging between .51 and .75). A similar pattern was found for the DZ correlations; studies with a small sample size showed more variance in the DZ correlations (with correlations ranging between –.14 to .54) than studies with larger sample sizes (with correlations ranging between .31 and .40).

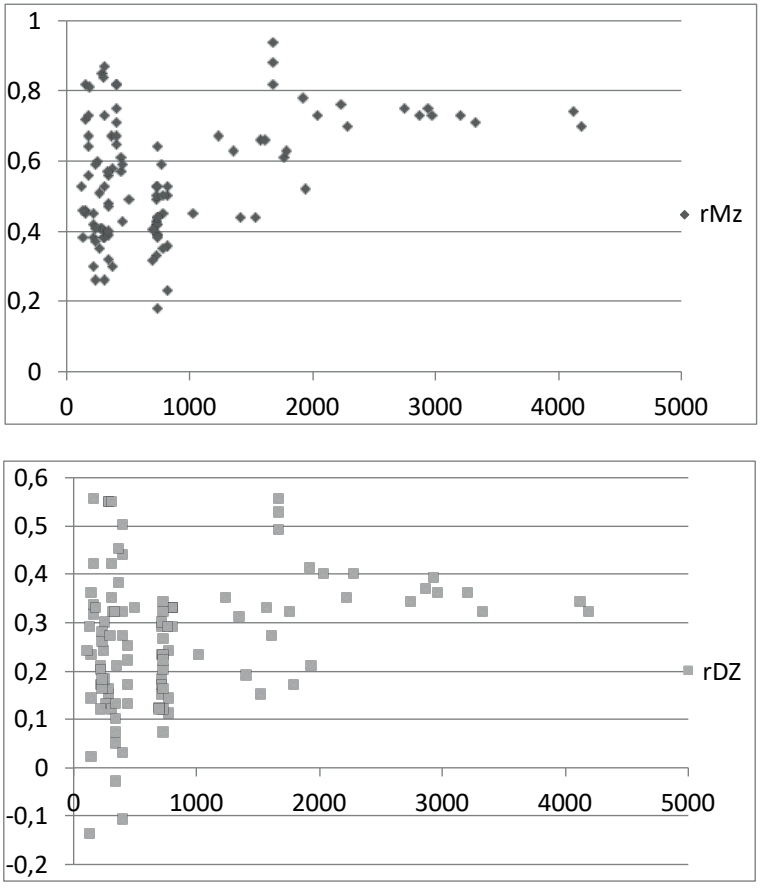

Figure 2. MZ correlations (above rMZ) and DZ correlations (below, rDZ) per sample size

Meta-analytic estimates

Meta-analyzing all data, applying multi-level analysis to take the dependency between effect sizes into account, resulted in an overall MZ correlation of 0.58 (ES_{Zmz} = .67, S.E. = .05, t = 13.27, p < .001, 95% CI = [.57, .77]) and an overall DZ correlation of .28 (ES_{Zdz} = .29, S.E. = .03, t = 9.48, p < .001, 95% CI = [.23, .34]). Applying Falconer's formula to calculate the heritability based on the meta-analytic MZ and DZ correlations results in an overall heritability of 60%. In other words, 60% of individual differences in self-control were due to genetic differences between people. The MZ correlation was twice as large as the DZ correlation, indicating little to no evidence for shared environmental effects. Rather, these results suggest that environmental effects on self-control, that explain 40% of the variance, are unique to individuals. This is in line with the standardized variance estimates reported by the studies, where 76% of the studies reported no or very little influence of the shared environment on the variance in self-control.

Next, we assessed whether the magnitude of the MZ or DZ correlation was moderated by study variables such as gender, age, and informant (see Table 2). Eleven papers tested for heritability differences in self-control between males and females, but none found significant differences between gender. This is confirmed by our moderator analyses, where gender did neither moderate the MZ correlations (F(1, 46)=.49, p=.49) nor the DZ correlations (F(1, 46)=.02, p=.90). This indicates that both the MZ and the DZ correlations are similar for males and females, indicating no differences in the heritability of self-control for males and females.

Important to note is that twin analyses examine differences in the variance, not differences in the mean. It could well be that males and females differ in their average self-control performance (see for example Duckworth et al., 2015), however, the relative contributions of genetic and environmental influences are equal across gender.

Initially, age did moderate both the MZ and DZ correlations, with higher MZ and higher DZ correlations in middle childhood compared to the other age groups. Informant also significantly moderated the twin correlations, with significantly higher MZ correlations for parent-report compared to self-report and observations. However, taking a closer look at the data illustrated that age and informant were not independent: in early and middle childhood, most assessments were based on parent-reports while assessments in adolescence and adulthood were mostly based on self-reports. Accordingly, we applied multiple-moderator models including both age and informant in the same model to take this dependency into account (see Table 3).

This multivariate analysis indicated that age did not significantly moderate the MZ correlation (F(3, 85)=1.79, p=.15) nor the DZ correlation (F(3, 81)=1.70, p=.17). Informant remained a significant moderator for the MZ correlations (F(2, 85)=17.00, p<.001), but not for the DZ correlations (F(2, 81)=2.10, p=.13). This indicates that differences in twin correlations were driven by differences in informants, rather than differences in age. More specifically, the MZ correlations were significantly higher when assessed by parent reports than self-reports and/or observations. The DZ correlations were similar across informants. MZ and DZ correlations did not significantly differ across self-reports and observations. Translating this to standardized heritability estimates using Falconer's formula, the heritability of self-control was significantly higher when assessed by parent-report (75%) as compared to self-report (53%) or observations (41%).

Table 2. Results for the univariate moderator analyses

		MZ			DZ		
Moderator	**Categories**	**ES_{Zmz}**	**95% CI**	**rMZ**	**ES_{Zdz}**	**95% CI**	**rDZ**
Gender							
	Female	.65	[.47, .83]	.57	.25	[.16, .32]	.24
	Male	.69	[.51, .86]	.60	.25	[.16, .33]	.25
Age	Early childhood	.68	[.56, .79]	.59	.37	[.29, .46]	.36
	Middle childhood	.85	[.74, .95]	.69	.36	[.28, .44]	.34
	Adolescence	.58	[.48, .67]	.52	.23	[.16, .31]	.23
	Adulthood	.51	[.40, .62]	.47	.17	[.09, .25]	.17
Informant	Parent report	.91	[.83, .99]	.72	.36	[.29, .43]	.35
	Self-report	.48	[.41, .55]	.45	.18	[.11, .25]	.18
	Observation	.57	[.47, .67]	.52	.32	[.24, .40]	.31

Note: ESz= Fisher's Z score, MZ=monozygotic twins, DZ=dizygotic twins, and CI= confidence interval

Table 3. Results for the multiple moderator analyses

Moderator variables	ES_z (SE)	95% CI	*t*-Statistic	*p*-Value
MZ correlations				
Intercept (ref.)	.86 (.06)	[.74, .99]	14.15	<.01
Slope Middle childhood	.10 (.07)	[-.04, .23]	1.50	.14
Slope Adolescence	-.05 (.09)	[-.22, .13]	-0.51	.61
Slope Adulthood	-.08 (.10)	[-.28, .13]	-0.81	.42
Slope Self-report	-.34 (.08)	[-.50, -.19]	-4.40	<.01
Slope Observation	-.31 (.07)	[-.44, -.18]	-4.74	<.01
DZ correlations				
Intercept (ref.)	.40 (.06)	[.29, .52]	6.89	<.01
Slope Middle childhood	-.03 (.07)	[-.16, .11]	-0.40	.69
Slope Adolescence	-.10 (.08)	[-.26, .05]	-1.32	.19
Slope Adulthood	-.17 (.08)	[-.33,-.00]	-2.04	.05
Slope Self-report	-.09 (.06)	[-.21, .02]	-1.63	.11
Slope Observation	-.07 (.05)	[-.16, .03]	-1.39	.17

Note: ESz= Fisher's Z score, MZ=monozygotic twins, DZ=dizygotic twins, and CI= confidence interval, ref.= reference category is early childhood and parent-report.

DISCUSSION

In the present meta-analysis, we synthesized research on the heritability of self-control from different behavior genetic studies. The analyses included monozygotic and dizygotic twin correlations of 31 twin studies, reporting a total of 108 correlations, covering an age range from 1.20 to 33.30 years, with a total sample size of more than 30,000 individual twins. The results of the meta-analysis of the twin correlations indicate an overall monozygotic twin pair correlation of .58 and an overall dizygotic twin pair correlation of .28, resulting in a heritability of 60% for self-control. Thus, 60% of the variation in self-control is due to genetic variation between individuals in the population. Overall, this indicates that there is indeed a robust genetic effect on self-control.

Moderator analyses revealed that monozygotic and dizygotic twin correlations did not differ for males and females, indicating no gender differences in the heritability of self-control. This is consistent with earlier

research illustrating that gender differences in heritability for a broad range of behavioral, psychiatric, and health related phenotypes are rare (Vink et al., 2012). In the present study, age did not moderate the heritability estimates of self-control, and we found influences of the unique environment rather than influences of the shared environment. The absence of common environmental influences may be specific to self-control. This is in line with traits closely related to self-control, such as ADHD and persistence, where research finds no influence of the shared environment across the lifespan (Kan et al., 2012; Keller et al., 2005). However, the absence of the common environment could also be a result of confounding informant effects. Most studies in childhood are limited to parent reports, and more research including multiple informants (i.e. parent-, self- and teacher-report) at the same age is necessary to distill whether the absence of C is specific to self-control or the result of reporter effects (Bartels, Boomsma, Hudziak, van Beijsterveldt & van den Oord, 2007; Wesseldijk et al., 2016).

The heritability of self-control implies that individual differences between persons in their self-control performance is partly explained by genetic differences between these individuals. This means that an individual with a predisposition for low self-control may struggle with the regulation of thoughts, behaviors, and impulses while an individual with a genetic predisposition for high self-control may excel in dealing with self-control challenges, although both individuals are exposed to the same environment. These findings imply that the environment – peers, parents, teachers – should take into account such innate individual differences in people's self-control capacities.

While some researchers advocated for the ban of twin studies (e.g., Burt & Simons, 2014), the present study underscores the importance of a multidisciplinary approach, including a genetic perspective, to comprehensively understand the etiology of self-control. In contrast to the article by Burt and Simons (2014), an increasing line of work emphasizes the importance of genetic sensitivity in the development of self-control (Beaver et al., 2013; Bolger, Meldrum & Barnes, 2018), embracing twin models as complementary models (Barnes et al., 2014). With the present meta-analysis, we demonstrate the potential of twin studies and a genetic perspective to stimulate future research on the etiology of self-control. Incorporating a genetic perspective in addition to an environmental perspective paves the way for a better understanding of the causes and consequences of self-control and provides new opportunities to improve self-control prevention and intervention efforts (Boisvert, Wright, Knopik & Vaske, 2012; Finkenauer et al., 2018; Harold, Leve & Sellers, 2017).

Future studies

While the current study provides evidence for a robust influence of genetic factors, it does not provide specific information about which sets of genes explain individual differences in self-control. Future work may employ Genome Wide Association Studies (GWAS) in order to unravel the specific genes that are linked to self-control. Considering the wide variety of research groups assessing self-control, and the worldwide increasing collection of DNA, applying a GWAS is feasible.

Monozygotic twin correlations are twice as large as the dizygotic twin correlations, suggesting evidence for a role of the unique environment and little influence of the shared environment. The absence of shared environmental influences does not mean that parents do not play a role in the development of self-control of their children (Ayoub et al., 2018; Engelhardt, Church, Paige Harden, & Tucker-Drob, 2018). Rather, it implies that environmental factors that make individuals dissimilar play a more important role, such as individual perceptions of parenting that make twins dissimilar even though they are raised within the same family (Cecil, Barker, Jaffee, & Viding, 2012; Hannigan, McAdams, Plomin, & Eley, 2016). However, more research is needed in order to specifically distill what unique environmental effects create individual differences in children growing up in the same family (Plomin & Daniels, 2011).

Genetic and environmental influences are not mutually exclusive or additive per se, and future research is recommended to investigate how gene-environment correlations (rGE) and gene-environment interaction (G x E) influence individual differences in self-control (Krueger, South, Johnson, & Iacono, 2008; Plomin, DeFries, & Loehlin, 1977). Some recent work suggest the presence of passive rGE, where parents create a home environment that is influenced by their own self-control which, in turn, correlates with the (genetic predisposition of) self-control of their child (Bolger, Meldrum, Barnes., 2018; Bridgett et al., 2015). Additionally, adoption studies suggest evidence for evocative rGE, where children with inherited regulation problems evoke more parental harshness (Fearon et al., 2015; Harold, Leve, & Sellers, 2017; Klahr et al., 2017).

Distinct from rGE, G x E entails that the genotypes vary in their sensitivity to the environment. In twin studies, genotypic influences are estimated conditional upon environmental exposure: when there is no interaction, the influence of genetic factors should not differ in the different environmental exposures. For example, recent work shows that the heritability of ADHD was the same across socio-economic strata indicating an absence of G x E, yet more

research is needed to replicate these findings (Gould, Coventry, Olson, & Byrne, 2018). On a molecular level, attempts to unravel G x E effects mostly concern candidate gene studies, yet the reliability of such methods is strongly debated (Dick et al., 2015). However, recently developed methodological techniques to take into account both environmental and genetic factors (e.g., genomic SEM or G x E with polygenic risk scores, Grotzinger et al., 2018; Peyrot et al., 2014), provide interesting avenues for future research on gene-environment interplay.

The age of participants in the included twin studies ranged between 1.2 and 33 years, with most studies reporting on middle childhood (7-12 years) or adolescence (13-18 years). Extending this line of work to middle and late adulthood is an important issue for future work. First, genetic research shows that heritability estimates for traits closely related to self-control (i.e., ADHD and emotion regulation) decreases over the course of adulthood (Kan et al., 2013; Nivard et al., 2015). Future work could explore whether a similar trend appears when investigating the heritability of self-control across the lifespan. Second, recent work in the social sciences illustrates how life events more typical to adulthood, such as marriage, children and loss of a loved one, explain individual differences in self-control (Bleidorn, 2015; Pronk, Buyukcan-Tetik, Illiás, & Finkenauer, 2019). To expand our knowledge on the etiology of self-control, applying classical twin models and gene-environment interplay models in adulthood, would be of particular interest to gain the necessary insights in the underlying mechanisms explaining individual differences in self-control across the lifespan.

Limitations

There are several limitations of this study that should be noted. First, a heritability estimate depends on the included sample as it is based on the variance of that specific population (Boomsma et al., 2002). Considering that most of the included studies were conducted in the USA reporting on non-clinical samples, we should be cautious when generalizing our findings to other populations. Further work needs to be done to establish heritability estimates in more diverse populations. Second, a wide variety of measures were used to assess self-control, and it would be interesting to assess heritability separately for each measure. However, the number of studies using the same measure was low. Considering that parameter estimates are poor when the number of studies is below five, we could not assess the influence of specific measurement on the heritability of self-control. Duckworth & Kern (2011) analyzed the phenotypic

correlations between different self-control measures, indicating that self-control is a coherent but multidimensional construct. For future research, it would be interesting to investigate the genetic correlations between these measures, allowing to examine to what extend all these measures tap into the same underlying construct.

Third, not all studies reported twin correlations of the full model possibly generating bias in the overall heritability estimates. However, we assume this bias is small because only few studies reported correlations based on best fitting models. Fourth, not modelling gene by environment interaction (G x E) might bias heritability estimates of the included twin studies (Purcell, 2002). More specifically, an interaction between A and C would result in an upward bias of A, while an interaction between A and E would result in an upward bias of E. However, despite the increasing interest in genetic factors explaining self-control, there is little work thus far investigating the contribution of G x E to self-control using twin designs. Further work modelling G x E needs to be done in order to gain insights whether such mechanisms explain individual differences in self-control, and whether they bias its heritability estimates.

Concluding remark

The current study suggests that genes significantly contribute to individual differences in self-control: the heritability of self-control is 60%. This finding provides further evidence for the importance of considering genetic influences when aiming to understand the underlying mechanisms contributing to the development of self-control across the lifespan.

Chapter 5

Genetic and environmental influences on self-control: Assessing self-control with the aseba self-control scale

ABSTRACT

This study used a theoretically-derived set of items of the Achenbach System of Empirically Based Assessment (ASEBA) to develop the Achenbach Self-Control Scale (ASCS) for 7 to 16 year olds. Using a large dataset of over 20,000 children, who are enrolled in the Netherlands Twin Register, we demonstrated the psychometric properties of the ASCS for parent-, self- and teacher-report by examining internal and criterion validity, and inter-rater and test-retest reliability. We found associations between the ASCS and measures of well-being, educational achievement, and substance use. Next, we applied the classical twin design to estimate the genetic and environmental contributions to self-control. Genetic influences accounted for 64% - 75% of the variance in self-control based on parent- and teacher-report (age 7 to 12), and for 47% - 49% of the variance in self-control based on self-report (age 12 to 16), with the remaining variance accounted by non-shared environmental influences. In conclusion, we developed a validated and accessible self-control scale, and show that genetic influences explain a majority of the individual differences in self-control across youth aged 7 to 16 years.

Keywords: self-control; self-report; teacher-report; parent-report; ASEBA; heritability

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Self-control – the capacity to alter unwanted impulses and behavior, in order to bring them into agreement with internal and external standards – is consistently associated with thriving mental, social, and physical well-being among children and adults (de Ridder, Lensvelt-Mulders, Finkenauer, Stok, & Baumeister, 2012; Moffitt et al., 2011; Tangney, Baumeister, & Boone, 2004). A validated scale allowing for longitudinal assessments of self-control from childhood to adolescence is needed to advance investigations of its development. A self-control scale suitable for children and adolescents should take several issues into account. First, in studying children's development, it is important that the scale is reliable across different ages. Second, we should take into account that children develop across contexts. The school context is different than the home context, with different raters providing different information (Bartels et al., 2007), and thus afford access to different behavior and insights that may be diagnostic for self-control. It is therefore important that a scale is reliable across different informants because, on the one hand, different informants afford a richer assessment of self-control, and, on the other hand, inter-rater reliability ensures robust assessment of self-control when only one rater is available. In the present study, we propose a scale that takes these issues into account.

Why is self-control important in children? Self-control entails *the strengthening of a desired action* (e.g., concentrating on an assignment, finishing homework, paying attention during class), and the capacity to *suppress an undesired impulse* (e.g., suppress temper tantrums, avoid breaking rules at home, inhibit irritable behavior in the classroom; Tangney et al., 2004). Self-control allows children to regulate their emotions, thoughts, or behavior, and underlies many skills and competences necessary to become healthy and well-adjusted adults (de Ridder et al., 2012; Finkenauer, Engels, & Baumeister, 2005). For example, low self-control in early childhood is associated with less happiness, less compliance, poorer educational achievement, and with more oppositional and deviant behaviors, such as substance use in later life (Duckworth et al., 2014; Finkenauer et al., 2005; Moffitt et al., 2011).

So far, a wide variety of questionnaires have been used to assess self-control. Some researchers use self-contained questionnaires, others select specific items of existing questionnaires. For example, Moffitt et al. (2011) assessed self-control by composing a scale of items selected from different scales, such as their Dunedin Behavioral Ratings. Their assessment included items such as "emotionally labile", "brief attention to tasks", and "impulsive". Likewise, Hay (2006) and Turner (2002) used a scale drawing items from the

Child Behavior Checklist (Achenbach & Rescorla, 2001) in combination with items of other child behavior scales such as the Rutter Behavior Scale (Hogg et al., 1997) including items such as "temper tantrums", "has difficulty completing activities", and "cannot wait for things". While there is a clear overlap in items included in these aforementioned studies, these composites of items have not yet resulted in the validation of an internationally accessible and applicable self-control scale, a crucial step to improve our understanding of self-control among children in the future. In this research, we therefore investigate whether a theoretically-derived set of items, similar to the aforementioned items, of the Achenbach System of Empirically Based Assessment (ASEBA, www.aseba.com) can be used to assess self-control during childhood. The ASEBA is a worldwide, frequently used, multi-informant tool applied in both scientific research and clinical practice (Achenbach, 2014). Validating a self-control scale based on such items could have vast implications. There are multiple large population based registers (e.g. NTR, Tchad, CATSS, Generation R, TRAILS) with longitudinal ASEBA data readily available (Anckarsäter et al., 2011; Lichtenstein, Tuvblad, Larsson, Carlström, 2007; Ormel et al.,2012; Jaddoe et al., 2012) A validated ASEBA self-control scale (ASCS) allows to calculate a score for self-control in retrospect. This richness of available longitudinal data is unique, and would be difficult to become available if self-control were to be assessed from now onwards. Additionally, the ASEBA questionnaires have been translated in over 100 languages facilitating prospective cross-cultural studies. This offers novel and exciting opportunities to examine theoretical suggestions regarding the development of self-control.

The widely used ASEBA includes the Child Behavior Checklist (CBCL), the Youth Self-Report (YSR), and Teacher's Report Form (TRF), which were tailored for parents, children, and teachers, respectively (Achenbach & Rescorla, 2001; Achenbach et al., 2002; So et al., 2012; Nelson et al., 2001). The ASEBA questionnaires were developed to assess child maladaptive functioning, including syndrome scales such as anxiousness, depression, somatic problems and later also applied to assess dimensions of Autism and obsessive-compulsive disorder (Achenbach & Rescorla, 2001; Achenbach et al., 2002). They measure comparable constructs across ages with similar item content, allowing us to select items that meet the theoretical conceptualization of self-control and that overlap in item content with existing self-control scales (Moffitt et al., 2011; Tangney et al., 2004). We selected 8 items, similar in content across informants. The current study examines the psychometric properties of this 8-item scale. Depending on the informant, we call this scale the ASCS parent-

report, ASCS self-report, or the ASCS teacher-report. We refer to these questionnaires collectively as the ASEBA Self-Control Scale or ASCS.

As a first step in examining the psychometric characteristics of the ASCS, we established their internal consistency and examined its dimensionality. In the literature on self-control, the dimensionality has been subject to discussion, some arguing that self-control is a unidimensional construct (Piquero, MacIntosch, & Hickman, 2000; Tangney et al., 2004), while others suggest that it is multi-dimensional (Duckworth & Steinberg, 2015; Maloney, Grawitch, & Barber, 2012; Williams, Fletcher, & Ronan, 2007). In addition, we tested for criterion validity of the ASCS. Next, we examined associations between the ASCS and several relevant outcomes including well-being, educational achievement (i.e., school results in math, language, education level in high-school and classroom compliance, evaluated individually), and substance use (i.e., alcohol use, drunk prevalence, smoking, evaluated individually). (Duckworth et al., 2014; Finkenauer et al., 2005; Moffitt et al., 2011). We investigated the reliability of the ASCS by testing their test-retest reliability and inter-rater reliability. A number of studies investigate the stability of self-control over time. Some find evidence supporting stability (Beaver et al., 2013; Turner et al., 2002), while others find evidence on malleability (Burt, Sweeten, & Simons, 2014; Hay & Forrest, 2006; Turner & Piquero, 2002). Although increases with time and age (maturation) have been found (Casey, 2015), longitudinal studies have reported substantial stability of self-control (Henneke et al., 2014). In line with these results, we expected that self-control will predict his/her own self-control in the future to a certain extent. Specifically, we expected that a child's level of self-control, as assessed by the ASCS at age 7, predicts his/her levels of self-control at later ages. Furthermore, we expected the mother-, father-, self- and teacher-reports to be significantly correlated, indicating agreement between informants, thus addressing the ability of the ASCS to appropriately measure self-control across contexts and informants.

Next, we looked at the genetic and environmental sources of individual differences in self-control assessed with the ASCS and estimated the heritability as a function of age, informant, and sex using the classical twin design (Boomsma, Busjahn, & Peltonen, 2002). Previous twin studies demonstrate that self-control "runs in families" (Bridgett, Burt, Edwards & Deater-Deckard, 2015). Several small-scale studies using adolescent twin data from Add Health (http://www.cpc.unc.edu/projects/addhealth) examined the genetic and environmental contributions to the variance in self-control. These studies showed heritability estimates between 44% and 64% for adolescent

self-reported self-control, with the remaining variance accounted for by non-shared environmental factors, and no sex differences (Beaver, Wright, DeLisi, & Vaughn, 2008; Beaver et al., 2009; Boisvert, Wright, Knopik, & Vaske, 2013). These results are largely consistent with more recent adolescent twin studies, such as the study by Anokhin and colleagues (2011), which reported a heritability estimate of 51% for self-control in 14-year-olds, and a study by Li et al. (2014), which reported a heritability estimate of 58% for self-control in 15-year-olds. In both studies the remaining variance was is accounted for by non-shared environmental factors. Studies using parent-reports consistently show stronger genetic influences, with most heritability estimates ranging between 74% and 79% (Lemery-Chalfant, Doelger, & Goldsmith, 2008; Li, Chen, Li, & Li, 2014), and one estimate of 95% (Beaver et al., 2013). Thus far, however, twin studies on self-control included relatively small samples (ranging between 372 and 825 twin pairs), few tested sex differences, and none included informantion from father- or teacher-reports. This study adds to this line of research by analyzing data from a large group of same-sex and opposite sex twin pairs, collected by the The Netherlands Twin Register (NTR), providing heritability estimates for mother-, father-, teacher- and self-report of self-control, from age 7 to age 16. We also tested sex differences applying scalar and non-scalar sex limitaton models.

METHODS

Sample and procedure

The NTR was initiated in 1987 in the Netherlands, and follows twins from childhood to adulthood (for more details see van Beijsterveldt et al., 2013). The present study includes measures of the ASEBA-CBCL/ ASEBA-TRF based on mother-, father- and teacher-report of children assessed at aged 7, aged 10 and aged 12, and measures of the ASEBA-YSR based on self-reports in children aged 12, aged 14 and aged 16. Accordingly, we assessed the reliability and validity of the ASCS based on mother-, father- and teacher-report of children aged 7, aged 10 and aged 12, and measures of self-control based on self-reports in children aged 12, aged 14 and aged 16 (the scales are consistent across ASEBA measures). The current study includes data from 24,704 7-year-olds (50.3% girls), 19,589 9/10-year-olds (50.7 % girls), 16,436 12-year-olds (50.9 % girls), with 1,704 self-reports for 12-year olds (50.8% girls), 10,020 14-year-olds (57,6% girls) and 7,566 16-year-olds (59,9 % girls).

Participants with a disease or handicap that interfered severely with daily functioning were excluded (N= 500). For same sex twin pairs, zygosity was based on DNA polymorphisms (N=1,578) or blood markers (N=240). For the remaining same-sex twin pairs, zygosity was determined using parent-reported items on resemblance in appearance and confusion of the twins. In approximately 93% of the cases, zygosity was correctly classified by these items (Rietveld et al., 2000). For the main analyses, we included all teacher reports, with slightly more than half of the twins sharing the same teacher (age 7, 54%; age 10, 53%; age 12, 57% of the twins were rated by the same teacher).

Measures

ASEBA. The ASEBA assessment consists of standardized questionnaires, which are completed by parents (CBCL), children themselves (YSR), and/or teachers (TRF). These questionnaires are used to rate a child's competencies and problems in the past 6 months (for parent- and self-report), or in the past 2 months (for teacher-report). The response format of the items is a 3-point scale, with response options *Not true* (coded 0), *Somewhat or Sometimes True* (coded 1), and *Very True or Often True* (coded 2). The CBCL and TRF consist of 113 items and the YSR of 112. Subsets of items are summed to create syndrome scales such as social problems, anxious depressed, and somatic complaints (Achenbach & Rescorla, 2001).

ASCS. The ASCS is intended to measure self-control as defined by person's ability to control his or her impulses, alter his or her emotions and thoughts, and to interrupt undesired behavioral tendencies and refrain from acting on them (Muraven & Baumeister, 2000). To develop the ASCS, we followed a systematic scale development procedure for item selection. In this procedure, two subject matter experts independently assessed the relevance of each item of the ASEBA to the theoretical conceptualization of self-control (Muraven & Baumeister, 2000). A third reviewer independently screened all ASEBA items selecting those corresponding to items used in earlier self-control studies. To resolve disagreement, in-depth discussion followed based the theoretical literature (Muraven & Baumeister, 2000; de Ridder et al., 2012) and earlier studies including separate items to construct a self-control scale (e.g., in line with items selected by Cecil et al., 2011; Moffitt et al., 2011; Hay, 2006 & Turner, 2002). As a result, eight items were selected for the ASCS (see Table 1), with four items of the attention problem scale (item 4, 8, 41, 78), four items of the aggressive behaviour scale (item 86, 87, 95), and one item of the rule breaking behaviour scale (item 28).

We calculated the scale score given three or fewer missing item responses (Achenbach & Rescorla, 2001). In the case of one to three missing item responses, we used the person-based weighted score. Cases with more than 3 items missing were excluded (2%), not expecting to influence variables of interest considering their low prevalence. Conducting our analyses in the subsample of participants without any missing values yielded similar results. Originally, the ASEBA was set up so that higher sum scores reflect higher frequency of child problems. Extending this approach to the ASCS, higher scores on the ASCS correspond to lower overall levels of self-control. This is in line with earlier studies on self-control (Moffitt et al., 2011).

Table 1. ASEBA Items included in the ASCS

Nr.	Item
4	Fails to finish things he/she starts
8	Can't concentrate, can't pay attention for long
28	Breaks rules at home, school, or elsewhere
41	Impulsive or acts without thinking
78	Inattentive or easily distracted
86	Stubborn, sullen, or irritable
87	Sudden changes in mood or feelings
95	Temper tantrums or hot temper

Note: Numbers are the same for parent, child and teacher reports, and based on the latest ASEBA instruments

Well-being. Well-being was assessed using the Cantril ladder (Cantril, 1965). Parents (age 7,9/10,12) and children (14,16) rated well-being on a ten-step 'ladder', with the bottom 'step' of the ladder representing the worst possible life and the top 'step' indicating the best possible life. Teachers rated well-being of 7, 9/10, and 12-year old children on a 5-point scale, with response options ranging from *always or almost always unhappy* (coded 1), *more often unhappy than happy* (coded 2), *equally often happy as unhappy* (coded 3), *more often happy than unhappy* (coded 4), *almost always happy* (coded 5).

Conners' Parenting Rating Scale/Teacher Rating Scale – Revised. This widely used instrument assesses the severity of behavior problems of children in the past month (Conners, Sitarenios, Parker, & Epstein, 1998a,1998b). The short version consists of 27 items for parent-report and 28 items for teacher-report (reported for age 7, 9/10, 12). Items are rated on a 4-point Likert scale

ranging from 0 = *not true at all (never, rarely)*, 1= *a little bit true (so now and then*), 2= *quite true (often, regularly),* 3 = *very much true (very often*), where higher scores indicate more severe symptoms. Cronbach's alphas were in line with the Conner's manual, reporting Cronbach's alphas between 0.83 – 0.85 for oppositional behavior, Cronbach's alphas between 0.78 and 0.90 for inattention, and Cronbach's alphas between 0.78 and 0.87 for hyperactivity (Conners et al., 1998a, 1998b).

Educational achievement. Educational achievement was assessed through school results in math, language, learning problems, behaviour in class and education level in high school, evaluated separately. Parents rated children's math and language achievement (on a 5-point scale, higher scores reflecting higher grades: 1 = *fail*, 2 = *weak*, 3 = *pass*, 4 = *good*, 5 = *excellent*), and learning problems ("did your child ever have learning problems?", on a two-point scale, 1 = *no*, 2 = *yes*). Teachers rated compliance and task orientation of the child ("in comparison to the average student in your class, how compliant is he/she?", in comparison to the average student in your class , how task orientated is he/she", 7-point scale, 1 = *much less*, 2 = *less* , 3 = *a little bit less*, 4 = *average*, 5= *little bit more*, 6 = *more*, 7 = *much more*). Adolescents (aged 14, 16) rated their level of education. The Dutch school system divides education level according to three levels: VMBO (preparing students for vocational training), HAVO (preparing students to study at universities of applied sciences) and VWO (preparing students for university), also referred to as lower level (coded as 1), middle level (coded as 2) and higher level education (coded as 3), respectively.

Substance use. Adolescents (aged 14, 16) were asked how often they smoked (1 = *never*, 2= *I quit smoking*, 3= *I smoke once a week*, 4= *I smoke multiple times per week* 5 = *I smoke multiple times per day*), their amount of alcohol intake per day in the weekend (1 = *less than 1 glass*, 2= *1-2 glasses*, 3= *3-5 glasses*, 4= *6-10 glasses* , 5= *11-16 glasses*, 6= *17-20 glasses*, 6= *17-20 glasses*, 7 = *more than 20 glasses*), and whether they had ever been drunk (0 = *never*, 1 = *1-2 times*, 2 = *3-4 times*, 3= *5-6 times*, 4= *7-8 times*, 5= *9-10 times*, 6=*11-19 times*, 7 = *20-39 times*, 8 = *more frequent*).

Strategy of analyses

In order to examine psychometric properties of the ASCS, we tested internal consistency, dimensionality, criterion validity, inter-rater reliability, test-retest reliability, and heritability estimates. We used SPSS 21 (IBM Corp., 2012) and Mplus version 7 (Muthén & Muthén, 2012) and conducted the analyses separately in children aged 7, 9/10, 12, 14, and 16, and

separately for mother-, father-, self- and teacher-report. To correct for the dependency of the observations due to clustering in families, a sandwich estimator was used with weighted least squares with mean variance adjusted (WLSMV) as the estimator (Rebollo, de Moor, Dolan, & Boomsma, 2012).

We investigated internal consistency by calculating Cronbach's alphas. The dimensionality was examined by fitting a Multimethod-Single trait confirmatory factor model (CFA) (Campbell & Fiske, 1959). This allowed us to establish whether the items measure a single factor (the single "trait" self-control) while taking into account the fact that the items are taken from different subscales within the ASEBA. In this manner, we can test the dimensionality of a model with one psychometric factor and multiple method/residual factors. Goodness of fit was evaluated using the Root Mean Square Error of Approximation (RMSEA), the Comparative Fit Index (CFI), and the Tucker Lewis Index (TLI). We adopted the rules of thumb that the RMSEA should be between .05 and .08 or lower (adequate fit in terms of error of approximation), and the TLI and CFI should be .95 or larger (Hu & Bentler, 1999).

We examined criterion validity by calculating cross-sectional and longitudinal correlations between ASCS and the variables mentioned above concerning adaptive behaviors (i.e., well-being, educational achievement and substance use). Additionally, we investigated inter-rater reliability by examining the correlations between the ASCS parent-, self- and teacher-report. We investigated test-retest reliability by investigating correlations between ASCS scores over time.

Next, we estimated the heritability of self-control in a classical twin design in Mplus version 7 (Muthén & Muthén, 2012). This design is built on the premise that differences in the resemblance between monozygotic twins (sharing approximately 100% of their DNA) and dizygotic twins (sharing 50% of their segregating genes on average) can be used to parse phenotypic trait variance into environmental and genetic components. As such, this model can be applied to estimate additive genetic (A, additive effects of alleles at multiple loci), non-additive or dominance genetic (D), common environment (C, the part of the variance that is shared by members of family), and non-shared environment (E, the part of the total variance that is unique to a certain individual) effects. We used raw-data genetic structural equation modelling with maximum likelihood estimation to perform univariate model fitting analyses to estimate the contributions of A, D or C, and E.

We first fitted a saturated model to estimate the twin correlations with their 95% confidence intervals. Based on these twin correlations an ACE or an ADE model with parameters allowed to differ between boys and girls was fitted to the data. Nested submodels were compared by hierarchic $\chi 2$ tests. The $\chi 2$ statistic was computed by subtracting - 2LL (log-likelihood) for the full model from that for a reduced model (v2 = - 2LL1- (- 2LL0)). Given that the reduced model is correct, this statistic is $\chi 2$ distributed with degrees of freedom (df) equal to the difference in the number of parameters estimated in the two models (Δ df = df1 - df0). In addition to the $\chi 2$ test statistic, Akaike's Information Criterion (AIC = v2 - 2df) was computed to compare non-nested models. A lower AIC indicates a better the fit of the model to the observed data. Quantitative sex differences were tested by constraining the A, C/D, and E parameters to be equal across sex. Based on the twin correlations, we see little support for qualitative sex differences, which were therefore not modelled. When sex differences appeared to be significant, a scalar-sex limitation model was tested. In this model, a difference in total variance between boys and girls is allowed, but the relative contributions of genetic and environmental influences are equal across gender. In order to test the significance of A, C/D factors, we fitted models without the parameter with confidence intervals including zero.

RESULTS

Internal consistency

Descriptive statistics of the ASCS are presented in Table 2. Cronbach's alpha coefficients suggested adequate internal consistency, with coefficients ranging between .81 and .83 for ASCS parent- and teacher-reports, between .70 and .73 for ASCS self-reports.

Table 2. Descriptives of ASCS including means, standard deviations and sample size for each rater (mother, father, teacher, self) and age (7 tot 16)

					Sample Size				
Age	**α**	**Informant**	***M***	***SD***	**MZM**	**DZM**	**MZF**	**DZF**	**DOS**
7	0.82	Mother	3.46	3.10	2050	2075	2286	1906	3871
7	0.81	Father	3.06	2.90	1453	1482	1671	1300	2684
7	0.81	Teacher	2.01	2.71	881	887	992	802	1631
10	0.82	Mother	3.33	3.11	1636	1572	1867	1463	3182
10	0.82	Father	2.87	2.90	1150	1083	1299	987	2161
10	0.82	Teacher	2.15	2.87	813	770	912	705	1559
12	0.82	Mother	2.95	2.93	1411	1337	1600	1274	2676
12	0.83	Father	2.64	2.83	988	938	1142	899	1859
12	0.82	Teacher	1.88	2.69	633	608	798	560	1135
12	0.73	Self	4.41	2.96	172	157	197	144	182
14	0.73	Self	4.27	2.76	739	670	1103	837	1661
16	0.70	Self	4.36	2.69	565	461	868	666	1223

Dimensionality

The ASCS consists of 8 items, with items derived from the attention problem scale (4 out of 10), aggressive behavior scale (3 out of 18), and rule-breaking behavior scale (1 out of 17). We specified a confirmatory factor model with one psychometric (trait, denoted SC) common factor representing self-control. In addition, we included one residual factor to account for the fact that the 4 items were taken from the attention problem scale ($R1_{att}$) and a second residual factor ($R2_{agg}$) to account for the fact that three items were taken from the aggressive behavior scale (see Figure S1, supplemental material). Statistically, this model showed good model fit for parent- and teacher-reports across all ages (see Table S2 and Table S3, supplemental material), supporting the psychometric unidimensionality of the scale.

For self-reports among adolescents aged 12 to 16 years, a correlation was added between the residuals of item 8 and item 78 ("can't concentrate/ can't pay attention for long", "inattentive, easily distracted"), because these items correlated highly. Given this addition, the model fitted well (see Table S2 and Table S3, supplemental material). The high correlation between these items suggests that children might have more difficulties making a distinction between the subtle meaning of these 2 items than adults, making children more likely to rate them more similarly (see Table S3, supplemental material).

Criterion validity

Consistent with the literature (e.g., Moffitt et al., 2011), cross-sectional associations between the ASCS and several relevant outcomes, such as well-being and educational achievement, were significant in the predicted directions (see Table 3). For example, low self-control at age 7 based on mother-report was significantly correlated with mother-rated Conners' oppositional behavior (.67), more learning problems (.28), and lower well-being (-.36). Similarly, low self-control at age 7 based on teacher-report was significantly correlated with teacher-rated Conners' oppositional behavior (.58), and lower well-being (-.35). It also correlated negatively with compliance (-.55), and task orientation in class (-.65), two measures that were unique to teacher-reports. These results replicated in cross-sectional correlations across ages and informants (see Table 3 for details, and Table S4 for the descriptives of measures included in tests of criterion validity).

In addition to the cross-sectional analyses, the self-control at age 7 was significantly correlated with constructs to which it should theoretically be related to at age 12 (see Table S4, supplemental material) and age 16 (see Table 4). For example, teacher-reported low self-control at age 7 was negatively correlated with self-reported education level at age 16 (-0.24). Mother-, father-, and teacher-reported low self-control at age 7 were positively and significantly correlated with self-reported smoking behavior at age 16, but were not significantly correlated with self-reported alcohol intake at age 16. Self-reported low self-control at age 14 was significantly correlated with both self-reported smoking and alcohol intake at age 16. See S6, supplemental material, for descriptives of measures included in criterion validity tests.

Table 3. Cross-sectional correlations between low self-control and validation constructs

Age	Informant	OP	IN	HYP	WB	LP	MA	LA	CO	TO
7	Mother	0.67	0.63	0.64	-0.36	0.28	-0.22	-0.25		
	Father	0.61	0.60	0.57	-0.34	0.26	-0.21	-0.23		
	Teacher	0.58	0.55	0.70	-0.35				-0.55	-0.65
10	Mother	0.69	0.64	0.64	-0.39	0.28	-0.24	-0.26		
	Father	0.64	0.63	0.60	-0.38	0.27	-0.21	-0.22		
	Teacher	0.62	0.49	0.71	-0.43				-0.57	-0.63
12	Mother	0.66	0.66	0.58	-0.35	0.30	-0.27	-0.29		
	Father	0.65	0.66	0.56	-0.33	0.29	-0.25	-0.28		
	Teacher	0.64	0.53	0.69	-0.32				-0.57	-0.60

Note. All correlations were significant at α <.001. Validation constructs include oppositional (OP), inattention (IN), hyperactivity (HYP), well-being (WB), learning problems (LP), school results math (MA), school results language (LA), compliance (CO) and task orientation (TO). LP, MA and LA were unique to parent reports, hence no correlations for teacher reports and these variables. CO and TO were unique to teacher reports hence no correlations for parent reports and these variables.

Table 4. Longitudinal correlations between low self-control at age 7, 12, and 14 and validation constructs at age 16, for mother-, father-, self- and teacher-reports

Age	Informant	SM	WB	AL	DR	EL
7	Mother	0.09*	-0.08*	0.04 ns	0.00 ns	-0.16
	Father	0.05*	-0.09*	0.01 ns	-0.03 ns	-0.15
	Teacher	0.11	0.01ns	0.03 ns	0.00 ns	-0.24
12	Mother	0.13	-0.11	0.08	0.03 ns	-0.28
	Father	0.12	-0.12	0.07	0.01 ns	-0.28
	Teacher	0.19	-0.05 ns	0.11	0.05 ns	-0.34
14	Self	0.21	-0.19	0.10	0.17	-0.09*

Note. ^{ns} not significant, *α <.01, all other correlations significant at α <.001. Validation constructs include smoking (SM), well-being (WB) , alcohol-use (AL), drunk prevalence (DR) and education level (EL).

Test-retest reliability & Inter-rater reliability

Inter-rater reliability of the ASCS was assessed by correlating ASCS measures over raters and time (see Table 5). Results showed significant cross-sectional correlations between the informants, with (a) significant and strong correlations between father- and mother- reports (.66 – .67), (b) significant and moderate correlations between parent- and child-reports (.40 – .44), (c) significant, but lower correlations between teacher- and self-reports (.29), and (d) significant and moderate correlations between teacher- and parent-reports (.32 – .40).

Longitudinal correlations of the ASCS scales across informants (intervals of 3 to 5 years) showed similar results, with (a) significant and moderate/strong correlations between father- and mother- reports (.45 – .67), (b) significant and small/moderate correlations between parent- and child-reports (.16 – .38), (c) significant and small correlations between teacher- and self-reports (.08 – .25), and (d) significant and small/moderate correlations between teacher- and parent-reports (.23 – .35). These findings, higher correlations between mother and father but low to moderate correlation between parent and child, parallel the cross-informant correlations reported by Achenbach and colleagues (2001, 2002), and earlier cross-informant studies on self-control (Duckworth & Kern, 2011), confirming the inter-rater reliability of the ASCS.

To examine test-retest reliability, we investigated correlations between self-control scores measured across time within raters, with time intervals of 3 to 5 years (see Table 5). The results showed (a) significant and strong correlations between mother-reports from age 7 to 12 (.57 – .67) and significant and strong correlations between father-reports from age 7 to 12 (.52 – .65), (b) significant and moderate/strong correlations between teacher-reports from age 7 to 12 (.43 – .54) and, (c) significant and moderate/strong correlations between self-reports from age 12 to 16 (.35 – .55). These results are consistent with longitudinal correlations of earlier studies on self-control (e.g., Turner & Piquero, 2002).

Table 5. Correlations of the ASCS scales between raters (parent-, self- and teacher-report) and over time (7,10,12,14 and 16)

Age	Informant	#	1	2	3	4	5	6	7	8	9	10	11
7	Mother	1											
	Father	2	0.66										
	Teacher	3	0.36	0.32									
10	Mother	4	0.65	0.51	0.32								
	Father	5	0.51	0.60	0.30	0.67							
	Teacher	6	0.30	0.25	0.47	0.39	0.36						
12	Mother	7	0.57	0.45	0.32	0.67	0.54	0.36					
	Father	8	0.46	0.52	0.32	0.54	0.65	0.34	0.67				
	Teacher	9	0.26	0.23	0.43	0.35	0.30	0.54	0.40	0.37			
	Child	10	0.36	0.30	0.25	0.38	0.32	0.23	0.44	0.40	0.29		
14	Child	11	0.23	0.20	0.13	0.24	0.21	0.16	0.31	0.28	0.24	0.41	
16	Child	12	0.17	0.16	0.08	0.18	0.16	0.11	0.23	0.21	0.15	0.35	0.55

Note. All correlations are significant at $\alpha < .001$.

Twin data modeling

Within-twin pair correlations of each zygosity group (MZM, DZM, MZF, DZF, and DZ opposite-sex) were inspected for initial exploration of the possible contribution of genetic and environmental factors (correlations are shown in Table 6). MZ correlations were high for all informants, with the size of the correlations being relatively stable for both males and females by parent-, teacher- and self-reports. These were approximately .70 - .75 for mother-report, .70 - .78 for father-report, .61-.67 for teacher-report, and .40 - .57 for self-report. This stability suggests that parent-, teacher- and self-report continue to report self-control in a fairly reliable way. MZ correlations were more than twice the DZ correlations at almost all ages and all informants, except for father report at ages 7, 10, and 12 and self-reports at age 12. Thus, one would expect genetic dominance or sibling interaction to be important for mother, teacher and self-reports. We observed no statistically significant zygosity effect on the variances in our data therewith suggesting the presence of D, rather than presence of a sibling interaction effect (Eaves, 1976; Rietveld, Posthuma, Dolan, & Boomsma, 2003).

Subsequently, we fitted a series of models that tested for relative contribution of additive genetic (A), dominant genetic (D) or common environment (C), and unique environmental (E) influences. First, we fitted ACE and ADE models separately allowing parameters to be estimated freely across sex. Based on

the lowest AIC value, we selected the best fitting model, that is an ACE or an ADE model. Second, to assess sex differences we fitted a model constraining parameters to be equal across sex. Third, when sex differences appeared, a scalar sex-limitation model was tested, allowing total variance to differ between boys and girls. Fourth, if confidence intervals of the estimated parameters included zero, we refitted the model dropping that specific parameter.

The best fitting models for mother- and teacher-ratings were ADE models with scalar sex-limitation. For self-report at age 14 and 16, an ADE model without sex differences showed the best model fit. A slightly different set of candidate models emerged for father report (age 7, 10, 12) and self-report (age 12) analyses. Comparing the AIC of an ADE and ACE model, an ACE model showed better model fit. However, the confidence intervals of C included zero, suggesting an AE-scalar model to be the final best fitting model for father-report (all ages) and an AE no sex-differences model for self-report (age 12) (see Table S6, Supplemental material, with the data presented by informant, age and model). Important to note is that we had a limited sample size at age 12 (*N*=1,704), so it is possible that we did not have enough power to detect D at this specific age (Rietveld et al., 2003).

The standardized estimates of the best fitting models are presented in Figure 1, and a full overview of estimates of the contributions of genetic and environmental factors are included in Table S7 of the supplemental material. For mother-, father- and teacher-report (age 7 to age 12), genetic influences accounted for 64% - 75% of the variance, with unique environmental influences accounting for 25% - 36% of the variance in self-control. For self-report, genetic influences accounted for 47% - 50% of the variance in self-control age 12 to age 16, with the remaining variance attributed to unique environmental factors. These estimates are in line with heritability estimates of earlier research (Beaver et al., 2009; Boisvert et al., 2013).

Table 6. Twin correlations (95% confidence intervals) for self-control age 7 to age 16 and across informants (mother-, father-, teacher- and self-report)

Age	Informant	MZM	DZM	MZF	DZF	DOS
7	Mother	0.74 (0.72 - 0.76)	0.34 (0.30 - 0.38)	0.70 (0.68 - 0.72)	0.32 (0.28 - 0.36)	0.31 (0.28 - 0.34)
	Father	0.75 (0.72 - 0.77)	0.39 (0.32 - 0.43)	0.73 (0.71 - 0.76)	0.36 (0.31 - 0.41)	0.32 (0.29 - 0.36)
	Teacher	0.61 (0.57 - 0.66)	0.32 (0.25 - 0.38)	0.63 (0.59 - 0.67)	0.17 (0.10 - 0.25)	0.27 (0.23 - 0.32)
10	Mother	0.73 (0.71 - 0.76)	0.36 (0.31 - 0.40)	0.71 (0.69 - 0.73)	0.32 (0.27 - 0.37)	0.32 (0.29 - 0.35)
	Father	0.76 (0.74 - 0.79)	0.35 (0.30 - 0.41)	0.70 (0.67 - 0.73)	0.40 (0.32 - 0.45)	0.31 (0.27 - 0.35)
	Teacher	0.66 (0.62 - 0.70)	0.33 (0.26 - 0.39)	0.66 (0.62 - 0.70)	0.27 (0.18 - 0.35)	0.22 (0.17 - 0.28)
12	Mother	0.75 (0.72 - 0.77)	0.34 (0.29 - 0.39)	0.73 (0.71 -0.75)	0.37 (0.32 - 0.42)	0.32 (0.29 - 0.36)
	Father	0.78 (0.76 - 0.81)	0.41 (0.36 - 0.47)	0.73 (0.70 - 0.75)	0.40 (0.35 - 0.46)	0.35 (0.31 - 0.39)
	Teacher	0.67 (062 - 0.72)	0.35 (0.27 - 0.43)	0.63 (0.59 - 0.68)	0.31 (0.21 - 0.41)	0.27 (0.20 -0.33)
	Self	0.57 (0.47 - 0.68)	0.32 (0.18 - 0.47)	0.40 (0.27 - 0.52)	0.32 (0.16 - 0.47)	0.03 (-0.13 - 0.19)
14	Self	0.44 (0.37 - 0.50)	0.19 (0.11 - 0.27)	0.52 (0.47 - 0.56)	0.21 (0.14 - 0.28)	0.16 (0.11 - 0.22)
16	Self	0.45 (0.38 - 0.53)	0.23 (0.13 - 0.34)	0.44 (0.38 - 0.50)	0.15 (0.05 - 0.24)	0.20 (0.13 - 0.28)

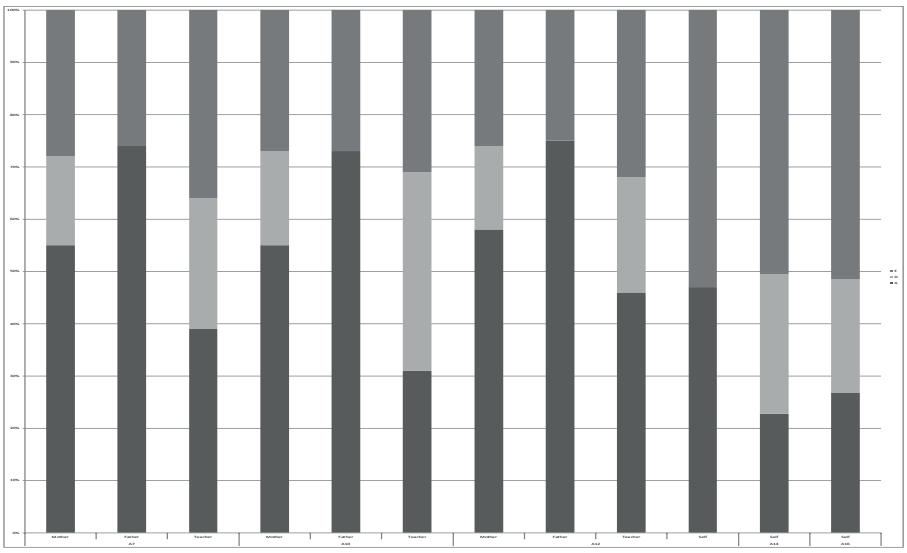

Figure 1. Estimates of relative contributions of genetic and environmental factors to self-control based on the best fitting model, for age 7, 10, 12, 14 and 16, for mother-, father-, teacher- and self-report, respectively.

DISCUSSION

This study reports the construction of a self-control scale for children and adolescents, the ASCS, using the existing item pool in the widely used ASEBA questionnaires. Strengths of the study include the capitalization on widely available items to measure self-control, the use of a very large sample, the analysis of the heritability of self-control, and the examination of multiple aspects of the new scale's psychometric functioning. The ASCS showed high internal consistency. In addition, we found high cross-sectional and longitudinal correlations between the ASCS and outcomes that were derived from the existing literature as being related to self-control (de Ridder et al., 2012; Moffitt et al., 2011), including well-being, educational achievement, and substance use. We also found that mother-, father-, self-, and teacher-reports were significantly correlated over time.

Adding to the psychometric soundness of the ASCS, we found heritability estimates paralleling earlier twin studies on self-control. A remarkable finding was that at age 12, genetic influences based on parent-reports accounted for

74% - 75% of the variance, while genetic influences based on self-reports accounted for only 47% of the variance. This pattern with higher heritability estimates for parent-reports than for self-reports has been reported by earlier studies on self-control and is a robust finding in the behavioral genetic literature (Anokhin et al., 2011; Beaver et al., 2009; Kan et al., 2014; Lemery-Chalfant et al., 2008). A body of empirical research attributes this finding to informant dependency; one important distinction between parent- and self-reports is that there is a single informant rating both twins (i.e., parent) versus different informants rating each twin (i.e., self-reports) (Bartels, Boomsma, Hudziak, van Beijsterveldt & van den Oord, 2007; Kan et al., 2014). However, the large genetic influence on self-control is in contrast to many non-genetic studies emphasizing the role of the 'common' environment rather than suggesting the role of genetics in the etiology of self-control, or the dynamical interaction between genes and environment. (cf. de Ridder et al., 2011; Pratt & Cullen, 2000). This shows the need to bridge results from behavioral geneticists and developmental psychologists in order to investigate the underlying mechanisms of self-control development in childhood.

Gottfredson and Hirschi (1990) argue in their General Theory of Crime that self-control is formed in childhood and remains relatively stable over time (absolute levels of self-control may change over time, but an individual's self-control relative to peers will be stable). Researchers using twin designs confirm the relative stability of self-control (cf. Beaver et al., 2008; 2013). As such, researchers in developmental psychology as well as in behavior genetics emphasize the importance of assessing self-control in youth to make inferences about adult adjustment. This is well illustrated in the recent work of Caspi and colleagues (2016), who assessed whether childhood risks forecast problems in adulthood. They found that children with low self-control were more likely to belong to high-cost economic burden groups as adults (e.g., using social welfare, smoking, crime, hospital stays, excessive weight). Policy makers are keen to improve well-being of adults by investing in child interventions. The returns of such an investment depends on the effectiveness of such interventions, and on the extent to which childhood outcomes predict adult adjustment. The ASCS can be used to assess self-control in youth, and thereby possibly for the prediction of adult adjustment.

In addition, the ASCS provide opportunities for secondary data-analyses. Specifically, our scale can be used to measure self-control in existing datasets, which include the ASEBA, but no questionnaires assessing self-control. Thereby, the ASCS may facilitate new research initiatives within existing research

projects. For example, investigating the association between self-control and established psychopathologies or other dimensions of adult adjustment.

The results of the current study should be interpreted with some limitations in mind. While the present study has used a large population-based sample of Dutch twin youth (van Beijsterveldt et al., 2013), we recommend caution in generalizing our findings to other countries. An important next step therefore would be to replicate our findings in different populations. Vazsonyi and Belliston (2007) have investigated associations between family relationships, low self-control, and deviance across seven countries, reporting similar patterns across cultures. Furthermore, cross-cultural heritability studies for other childhood behavioral problems report on similar genetic architectures in different countries (cf. Porsch et al., 2016). Conducting comparable research would provide information on the cross-cultural validity and reliability of the ASCS. Replicating this study in twin data with a larger sample size of self-reports at age 12 is especially recommended, as research needs to clarify possible changes in environmental and genetic contributions to self-control from childhood to adolescence. Considering the international character of the ASEBA, and the wide variety of research groups including the ASEBA in their data collections, replicating this study is feasible.

Despite these limitations, the ASCS may provide insights and breakthroughs for longstanding questions and problems in the study of self-control, its links with adjustment and achievement across the lifespan, and its capacity for integration across multiple levels of analysis is especially high.

SUPPLEMENTS

CHAPTER 5

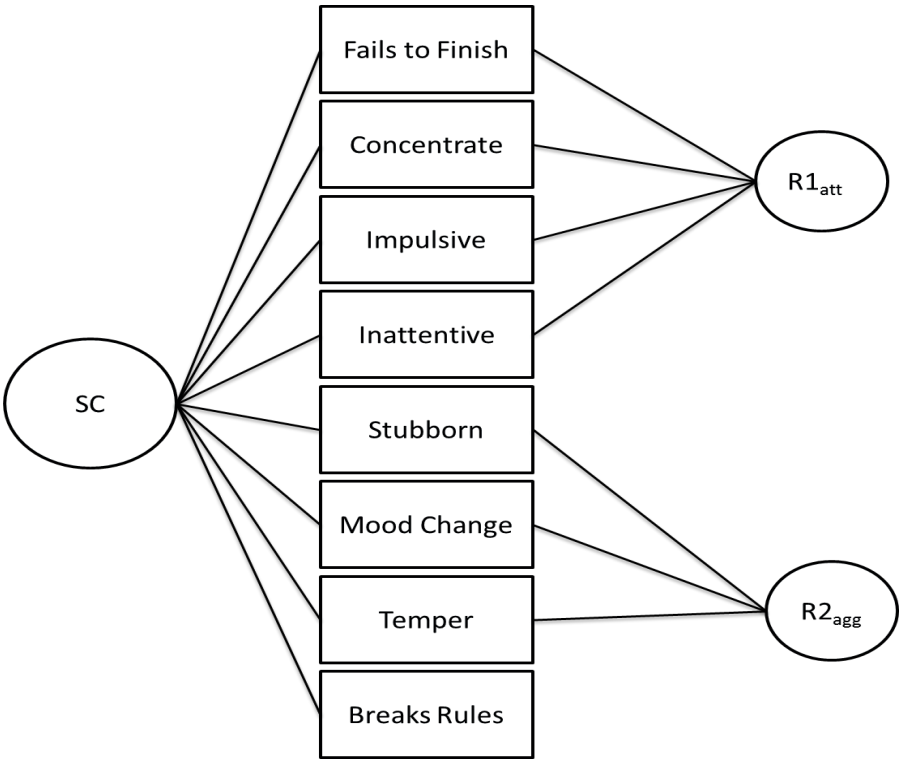

Figure S1. Dimensionality ASCS, with one psychometric factor (SC, self-control) and two residual factors ($R1_{att}$, $R2_{agg}$).

Table S2. Model fit indices of ASCS for mother(MR), father (FR), teacher and self-report (SR) from age 7 till 16

	A7			**A10**			**A12**				**A14**	**A16**
	MR	**FR**	**TR**	**MR**	**FR**	**TR**	**MR**	**FR**	**TR**	**SR**	**SR**	**SR**
RMSEA	0.01	0.01	0.02	0.02	0.02	0.02	0.02	0.03	0.02	0.02	0.03	0.04
CFI	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	0.99	1.00	0.99
TLI	1.00	1.00	1.00	1.00	1.00	1.00	1.00	0.99	1.00	0.99	0.99	0.99

Table S3. Standardized factor loadings of psychometric factor Self-Control from Confirmatory Factor Analyses from age 7 to 16 for mother (MR)-, father (FR)-, self (SR)- and teacher (TR)-report respectively, all are significant at α <.001

		A7									**A10**								
		MR			**FR**			**TR**			**MR**			**FR**			**TR**		
Nr.	Item Content	F	R1	R2	F	R1	R2	F	R1	R2	F	R1	R2	F	R1	R2	F	R1	R2
4	Fails to finish things he/she starts	.53	.51		.55	.50		.56	.56		.58	.43		.61	.46		.56	.55	
8	Can't concentrate	.53	.80		.52	.83		.56	.81		.56	.80		.56	.80		.56	.81	
41	Impulsive or acts without thinking	.69	.25		.69	.23		.78	.25		.74	.22		.70	.25		.83	.22	
78	Inattentive or easily distracted	.66	.60		.64	.56		.64	.64		.63	.60		.66	.58		.63	.65	
86	Stubborn, sullen, or irritable	.57		.60	.56		.62	.59		.65	.58		.62	.60		.58	.65		.60
87	Sudden changes in mood or feelings	.62		.51	.59		.55	.62		.60	.62		.55	.63		.51	.70		.52
95	Temper tantrums or hot temper	.60		.55	.60		.56	.69		.57	.62		.52	.62		.55	.71		.53
28	Breaks rules at home, school, or elsewhere	.80			.80			.89			.79			.81			.85		

		A12												**A14**			**A16**		
		MR			**FR**			**TR**			**SR**			**SR**			**SR**		
Nr.	Item Content	F	R1	R2	F	R1	R2	F	R1	R2	F	R1	R2	F	R1	R2	F	R1	R2
4	Fails to finish things he/she starts	.58	.43		.60	.45		.61	.52		.47	.56		.50	.39		.49	.27	
8	Can't concentrate	.57	.77		.60	.76		.58	.81		.44	.07		.47	.30		.41	.58	
41	Impulsive or acts without thinking	.70	.28		.70	.27		.83	.22		.63	.16		.68	.08		.65	.10	
78	Inattentive or easily distracted	.64	.61		.66	.57		.68	.60		.61	.35		.66	.24		.65	.18	
86	Stubborn, sullen, or irritable	.61		.59	.64		.56	.68		.54	.49		.56	.46		.44	.43		.45
87	Sudden changes in mood or feelings	.58		.56	.64		.53	.67		.59	.58		.26	.46		.40	.46		.34
95	Temper tantrums or hot temper	.66		.47	.64		.49	.74		.51	.57		.31	.54		.46	.52		.54
28	Breaks rules at home, school, or elsewhere	.81			.83			.85			.62			.62.			.58		

Table S4. Descriptives of measures included in tests of criterion validity

Age	Informant	OP	IN	HYP	WB	LP	MA	LA	CO	TO
7	Mother	3.95 (3.37)	3.08 (3.80)	2.75 (3.26)	8.41 (0.96)	1.18 (0.38)	3.69 (0.84)	3.60 (0.87)		
	Father	3.94 (3.29)	2.88 (3.52)	2.88 (3.17)	8.36 (0.93)	1.15 (0.36)	3.80 (0.82)	3.71 (0.84)		
	Teacher	0.64 (1.54)	2.01 (2.91)	1.98 (3.30)	1.57 (0.73)				4.51 (1.39)	4.79 (1.51)
10	Mother	3.95 (3.45)	3.50 (3.96)	2.24 (3.07)	8.33 (1.01)	1.28 (0.45)	3.74 (0.95)	3.63 (0.93)		
	Father	3.68 (3.33)	3.31 (3.77)	2.32 (2.95)	8.22 (1.01)	1.23 (0.42)	3.76 (0.90)	3.69 (0.87)		
	Teacher	0.80 (1.77)	2.55 (3.13)	1.81 (3.08)	1.71 (0.79)				4.60 (1.43)	4.84 (1.58)
12	Mother	3.53 (3.19)	3.04 (3.71)	1.53 (2.44)	8.25 (1.13)	1.31 (0.46)	3.79 (0.98)	3.63 (0.94)		
	Father	3.33 (3.17)	2.95 (3.59)	1.67 (2.49)	8.25 (1.02)	1.27 (0.44)	3.80 (0.95)	3.71 (0.91)		
	Teacher	0.81 (1.73)	2.33 (2.94)	1.39 (2.69)	1.69 (0.81)				4.82 (1.44)	5.15 (1.49)

Note. Validation constructs include oppositional (OP), inattention (IN), hyperactivity (HYP), well-being (WB), learning problems (LP), school results math (MA), school results language (LA), compliance (CO) and task orientation (TO).

Table S5. Longitudinal correlations between low self-control (LSC) at age 7 and validation constructs at age 12 for mother, father, teacher report

		Age 12								
Age	Informant	OP	IN	HYP	WB	LP	MA	LA	CO	TO
7	Mother	.46	.42	.43	-.23	.26	-.20	-.23		
7	Father	.36	.35	.35	-.17	.23	-.17	-.19		
7	Teacher	.17	.37	.26	-.11	.31	-.26	-.29	-.32	-.41

Note. All correlations are significant at $\alpha < .001$. Validation constructs include oppositional (OP), inattention (IN), hyperactivity (HYP), well-being (WB), learning problems (LP), school results math(MA), school results language (LA), compliance (CO) ,task orientation (TO).

Table S6. Number of participants (N) included in longitudinal correlations

		Age 16				
		SM	**WB**	**AL**	**DR**	**EL**
	***M* (*SD*)**	**1.46 (1.14)**	**7.71 (1.09)**	**2.61 (1.38)**	**1.87 (2.25)**	**1.95 (0.84)**
Age	Informant					
7	Mother	4646	4701	2063	4737	4725
7	Father	3899	3950	1745	3970	3977
7	Teacher	1729	1802	1175	1803	1801
12	Mother	4217	4277	1818	4299	4300
12	Father	3338	3378	1442	3404	3396
12	Teacher	2088	2093	932	2117	2118
14	Self	3168	3232	1918	3244	3246

Note. Validation constructs include smoking (SM), well-being (WB), education level (EL), alcohol-use (AL), drunk prevalence (DR).

Table S7. Model-fitting results of univariate genetic analyses of self-control (ASCS)

Age	Informant		Model	ep	-2LL	AIC	Comp	Δχ²	*p*
A7	Mother	0	Saturated	25	-58399.65	116849.30	-	-	-
		1	ACE sex diff	8	-58450.75	116917.50	0	51.10 (17)	<.001
		2	ADE sex diff	8	-58445.8	116907.61	0	46.15 (17)	<.001
		3	ADE no sex diff	5	-58527.69	117065.37	2	81.89 (3)	<.001
		4	**ADE scalar**	**6**	**-58454.72**	**116921.44**	**2**	8.92 (2)	**0.01**
	Father	0	Saturated	25	-39959.13	79968.26	-	-	-
		1	ACE sex diff	8	-39990.04	79996.08	0	30.91 (17)	0.02
		2	ADE sex diff	8	-39990.07	79996.15	0	30.94 (17)	0.02
		3	ACE no sex diff	5	-40053.70	80117.40	1	61.30 (3)	<.001
		4	ACE scalar	6	-39992.40	79996.80	1	2.36 (2)	0.31
		5	**AE scalar**	**5**	**-39992.40**	**79994.80**	**4**	0.00 (1)	**1.00**
	Teacher	0	Saturated	25	-22419.99	44889.97	-	-	-
		1	ACE sex diff	8	-22439.8	44895.6	0	19.81 (17)	0.28
		2	ADE sex diff	8	-22433.06	44882.11	0	13.07 (17)	0.73
		3	ADE no sex diff	5	-22579.03	45168.06	2	145.97 (3)	<.001
		4	**ADE scalar**	**6**	**-22435.42**	**44882.83**	**2**	**2.36 (2)**	**0.31**
A10	Mother	0	Saturated	25	-46708.73	93467.46	-	-	-
		1	ACE sex diff	8	-46740.68	93497.36	0	31.95 (17)	0.02
		2	ADE sex diff	8	-46735.26	93486.51	0	26.53 (17)	0.07
		3	ADE no sex diff	5	-46820.35	93650.69	2	85.09 (3)	<.001
		4	**ADE scalar**	**6**	**-46738.21**	**93488.43**	**2**	**2.95 (2)**	**0.23**
	Father	0	Saturated	25	-31180.7	62411.39	-	-	-
		1	ACE sex diff	8	-31200.72	62417.44	0	20.02(17)	0.27
		2	ADE sex diff	8	-31202.16	62420.31	0	21.46 (17)	0.21
		3	ACE no sex diff	5	-31249.66	62509.32	1	48.94 (3)	<.001
		4	ACE scalar	6	-31209.59	62431.17	1	8.87 (2)	0.02
		5	**AE scalar**	**5**	**-31209.60**	**62429.17**	**4**	0.01 (1)	0.92
	Teacher	0	Saturated	25	-20539.2	41128.41	-	-	-
		1	ACE sex diff	8	-20566.49	41148.99	0	27.29 (17)	0.05
		2	ADE sex diff	8	-20558.56	41133.11	0	19.36 (17)	0.31
		3	ADE no sex diff	5	-20793.16	41596.33	2	234.60 (3)	<.001
		4	**ADE scalar**	**6**	**-20561.24**	**41134.48**	**2**	**2.68 (2)**	**0.26**
A12	Mother	0	Saturated	25	-39092.18	78234.36	-	-	-
		1	ACE sex diff	8	-39128.34	78272.67	0	36.16 (17)	<.001

		2	ADE sex diff	8	-39125.55	78267.1	0	33.37 (17)	0.01
		3	ADE no sex diff	5	-39088.56	78187.11	2	36.99 (3)	<.001
		4	**ADE scalar**	**6**	**-39127.69**	**78267.39**	**2**	2.14 (2)	**0.34**
	Father	0	Saturated	25	-26849.6	53749.21	-	-	-
		1	ACE sex diff	8	-26863.74	53743.48	0	14.14 (17)	0.66
		2	ADE sex diff	8	-26864.86	53745.72	0	15.26 (17)	0.58
		3	ACE no sex diff	5	-26939.56	53889.11	1	75.82 (3)	<.001
		4	ACE scalar	6	-26869.6	53751.20	1	5.86 (2)	0.05
		5	**AE scalar**	**5**	**-26870.74**	**53751.49**	**4**	**1.14 (1)**	**0.29**
	Teacher	0	Saturated	25	-15339.62	30729.24	-	-	-
		1	ACE sex diff	8	-15369.48	30754.96	0	29.86 (17)	0.03
		2	ADE sex diff	8	-15367.5	30750.99	0	27.88 (17)	0.05
		3	ADE no sex diff	5	-15585.04	31180.08	2	217.54 (3)	<.001
		4	**ADE scalar**	**6**	**-15369.51**	**30751.01**	**2**	2.01 (2)	**0.37**
	Self	0	Saturated	25	-4020.12	8090.23	-	-	-
		1	ACE sex diff	7	-4023.85	8061.7	0	3.73 (18)	1.00
		2	ADE sex diff	7	-4026.3	8066.6	0	6.18 (18)	0.98
		3	ACE no sex diff	4	-4030.17	8068.32	1	6.32 (3)	0.10
		4	**AE no sex diff**	**3**	**-4030.20**	**8066.32**	**3**	0.03 (1)	0.86
A14	Self	0	Saturated	25	-21880.83	43811.65	-	-	-
		1	ACE sex diff	7	-21898.04	43810.07	0	17.21 (18)	0.51
		2	ADE sex diff	7	-21895.24	43804.47	0	14.41 (18)	0.70
		3	**ADE no sex diff**	**4**	**-21899.09**	**43806.18**	**2**	3.85 (3)	**0.28**
A16	Self	0	Saturated	25	-15281.66	30613.31	-	-	-
		1	ACE sex diff	7	-15309.90	30633.80	0	28.24 (18)	0.06
		2	ADE sex diff	7	-15307.75	30629.49	0	26.09	0.09
		3	**ADE no sex diff**	**4**	**-15309.88**	**30627.75**	**2**	2.13 (3)	**0.55**

Note: In **Bold** are the best fitting models.

Chapter 6

Investigating the association between family connectedness and self-control in adolescence in a genetically sensitive design

ABSTRACT

Objective: Family connectedness is key for the development of self-control in early and middle childhood. But is family connectedness still important during the transitional phase of adolescence, when adolescents demand more independence from their parents and rely more on their peers? The aim of the present study was to investigate the association between family connectedness and self-control, and whether it still holds in adolescence using a genetically sensitive design. **Method:** Data were used from a large sample of twins aged 14 (*N*=11,260) and aged 16 (*N*=8,175), all enrolled in the Netherlands Twin Register. We applied bivariate twin models and monozygotic twin difference models to investigate the association between family connectedness and self-control and to unravel to what extent genetic and environmental factors explain this association. **Results:** The results showed that more family connectedness is significantly related to better self-control in adolescence, albeit with a small effect size. Twin analyses revealed that this association was mainly explained by common genetic factors and that the effects of environmental factors were small. **Conclusions:** The current findings confirm the role of family connectedness in adolescent self-control. Importantly however, the results demonstrate that phenomena we see within families seem the product of parent and children sharing the same genes rather than being exclusively attributable to environmental processes.

Keywords: family connectedness; self-control; adolescence; twins; genetics; environment

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Family connectedness encompasses the feeling of trust, understanding, and support within the family, and is robustly associated with healthy child development (Chu, Saucier, & Hafner, 2010; Hagerty, Lynch-Sauer, Patusky, Bouwsema, & Collier, 1992). Children develop over time, and so does the influence of the family on the development of the child (Sameroff, 2010). This development is especially pronounced during adolescence, as adolescents increasingly become active agents in their own development, demand more independence from their parents, and rely more on connectedness with peers than on connectedness with the family (Casey & Caudle, 2013). The developmental transition from being dependent on parents to independence yields an important question: Is family connectedness still related to person characteristics in adolescence and, if so, what is the nature of this association? Thus far, most research examining this question focused on early and middle childhood rather than adolescence. Moreover, few studies take the possibility of genetic confounding into account. Unraveling the underlying genetic and environmental mechanisms is important to understand how adolescents develop within, and in interaction with, their social world (Harold, Leve, & Sellers, 2017; Plomin & Daniels, 2011).

Self-control

A key person characteristic in adolescent development is self-control. Self-control is the capacity to alter unwanted impulses and behaviors in order to bring them into agreement with internal and external standards (Duckworth & Steinberg, 2015; Gillebaart, 2018). Self-control is proposed to be especially important during adolescence, as adolescents with high self-control capacities have higher quality interpersonal relationships, better school grades, healthier lifestyles, less psychological problems, and report more happiness than their adolescent peers with less self-control capacities (Caspi et al., 2016; Finkenauer, Engels, & Baumeister, 2005; Moffitt et al., 2011). Given these findings, self-control has also been coined a hallmark for adolescents to become well-adjusted adults (Casey & Caudle, 2013; Caspi et al., 2016; Moffitt et al., 2011). As such, it is important to understand causes and consequences of individual differences in adolescent self-control capacities.

The association between family connectedness and self-control

Research in early and middle childhood shows that family connectedness and self-control are associated. When parents create a context where family members feel accepted and supported, children get the opportunity to learn how to self-regulate their behaviors and impulses (Bowlby, 1969; Pallini et al., 2018). Additionally, family connectedness is closely related to higher-quality parenting which, in turn, affects children's opportunities to learn how to self-regulate their impulses, behaviors, and emotions (Sheffield Morris, Silk, Steinberg, Myers, & Robinson, 2007). Importantly, the association between family connectedness and self-control can also be explained by a child's level of self-control evoking certain family responses. For example, children with high self-control elicit trust, warmth, and affection from their parents and siblings (Buyukcan-Tetik, Finkenauer, Siersema, Vander Heyden, & Krabbendam, 2015), feelings which strengthen family connectedness (Tiberio et al., 2016). Thus, there seems to be a reciprocal association between family connectedness and a child's self-control.

Research in adolescence on the association between family connectedness and self-control is more inconclusive. For example, while some longitudinal studies in adolescents find no significant bidirectional association (Craig, 2016; Moilanen, Rasmussen, & Padilla-Walker, 2015), others find significant associations with small effect sizes (Janssen, Eichelsheim, Dekovic, & Bruinsma, 2016; Tiberio et al., 2016), and again others find medium effect sizes for the association between family connectedness and self-control (Hay, Meldrum, & Piquero, 2013).

A particular problem in the current literature is that the associations reported in the studies are commonly interpreted as reflecting causal effects between family connectedness and self-control. Most of these studies, however, are correlational and thereby do not necessarily provide evidence of true direction of effect. An alternative explanation for the association between family connectedness and self-control is that underlying factors influence them both (i.e., no direct causal relationship but a third underlying factor that drives the association between the two). One key underlying factor may be genetic factors.

Genetic influences on family connectedness and self-control

Over the last decade, accumulating research shows that traits are at least partly heritable (Polderman et al., 2015). For example, a recent meta-analysis showed that individual differences in self-control are for 60% explained by

genetic differences between people (Willems et al., 2019a). Traits closely related to self-control show similar heritability estimates such as grit (37%, Rimfeld, Kovas, Dale, & Plomin, 2016), effortful control (49%, Yamagata et al., 2005), delay discounting (51%, Anokhin, Golosheykin, Grant, & Heath, 2011), and attention skills (70%, Polderman et al., 2007). Importantly, not only person characteristics, but also contextual factors are partly influenced by genetic factors. Genes do not in any direct way "code" people for specific environments, however, individual's genetic make-up influences their perception and selection of contexts (Plomin & Daniels, 2011). Individual differences in family connectedness are for 30% to 40% influenced by genetic factors (van der Aa, Boomsma, Rebollo-Mesa, Hudziak, & Bartels, 2010), and the way children perceive parenting is correlated with their genes (gene-environment correlation) (Hannigan, McAdams, Plomin, & Eley, 2016; Harden et al., 2007; D'Onofrio et al., 2007).

Considering the genetic contribution to both family connectedness and self-control, it may thus well be that their observed associations are explained by common genetic factors that simultaneously influence both family connectedness and self-control (Pingault et al., 2018; Ayoub et al., 2019). Thus far, however, studies on the association between family connectedness and self-control specifically applying genetically sensitive designs are scarce. One study using a genetic sensitive design found no significant association between parental socialization and self-control in adolescence after controlling for genetic influences. Yet, the study had limited statistical power to solidify these assumptions (Monozygotic twin pairs N=289, Dizygotic twin pairs N= 452). To further our knowledge it is important to assess whether family connectedness and self-control are the result of a true directional effect or, alternatively, by a confounding third factor such as common genetic factors simultaneously influencing both the family context and child outcomes (genetic pleiotropy, Pingault et al., 2018).

One design allowing researchers to investigate the association between family connectedness and self-control in adolescents, while simultaneously unraveling to what extent the association is influenced by genetic or environmental factors, is the bivariate twin design. This design is built upon the premise that monozygotic twin pair correlations (100% genetically identical) and dizygotic twin pair correlations (on average 50% genetically identical) can be parsed into environmental and genetic influences (Boomsma, Busjahn, & Peltonen, 2002).

Additionally, twin data allow researchers to apply a monozygotic difference designs (Bartels, de Moor, van der Aa, Boomsma, & de Geus, 2012; Cecil, Barker, Jaffee, & Viding, 2012; De Moor, Boomsma, Stubbe, Willemsen, & de Geus, 2008). If there is a causal relationship between family connectedness and self-control, it could be expected that in genetically identical twins, the twin who perceives more family connectedness has higher self-control than his/her co twin, or vice versa. Applying both twin designs, the bivariate twin design and the monozygotic difference design, thereby allows us to better understand the nature of the association between family connectedness and self-control.

Current study

Previous studies have mainly focused on the association between family connectedness and self-control in middle and early childhood. We aim to extend this line of work to adolescence, a transformative phase for families, parents, and children. Additionally, few studies thus far have investigated the association between family connectedness and self-control in a genetically sensitive design. Such a design can provide information on the extent to which environmental or genetic factors influence this association. The goal of this study is therefore to investigate the nature of the association between family connectedness and self-control. We aim to do so by investigating the following three sub-questions in a large longitudinal sample of adolescent twins aged 14 years and aged 16 years: 1) Are family connectedness and self-control associated over the course of adolescence? 2) To what extent is the association explained by genetic or environmental influences? 3) Can we determine the directionality of the association between family connectedness and self-control?

METHOD

Sample and procedure

Longitudinal survey data were collected in twins registered with the Netherlands Twin Register, a population-based study initiated in 1987 in the Netherlands at the Vrije Universiteit Amsterdam. Upon parental consent, 14- and 16-year-old twins received questionnaires on family functioning, physical health, and psychological well-being (see van Beijsterveldt et al., 2013 and van der Aa et al., 2010 for more details on data collection). Data collection was approved

by the Medical Ethical Committee at the Vrije Universiteit Medical Center (2003/182).

The dataset comprised of 14-year-old twins (57.6% females, MZ twin pairs *N*=1,905, DZ twin pairs *N*=3,353), and 16-year old twins (58.1% females, complete twin pairs: 84%, MZ twin pairs *N*=1,483, DZ twin pairs *N*=2,476). For 28.1% of the same-sex twin pairs, zygosity was determined based on DNA typing or blood group. For the remaining same-sex twins, zygosity was determined based on questionnaire items filled in by parents (e.g. "is it difficult to discern the two siblings from one another"), resulting in accurate determination of zygosity in 93% of the cases (Rietveld et al., 2000).

Measures

Family connectedness was assessed with an adolescent self-report subscale of the McMaster Family Assessment Device (FAD, Epstein, Baldwin, & Bishop, 1983). We used the Dutch translation which shows good psychometrical properties (van der Aa et al., 2010; Wenniger, Hageman, & Arrindell, 1993), a one factor structure, and a Cronbach's Alpha coefficient of .84 at age 14 and .85 age 16. The subscale consisted of 6 items tapping into family connectedness, such as "in times of crisis, we can turn to each other for support", "we feel free to express our feelings within the family", and, "we trust each other". Items were scored on a 4-point scale, ranging from 1= *Strongly Disagree* to 4= *Strongly Agree*. Scores on individual items were summed, so that higher scores reflected more family connectedness.

Self-control was assessed with the adolescent self-report of the ASEBA Self-Control Scale (ASCS, Willems et al., 2018a). This scale shows to be psychometrically sound to assess self-control (Willems et al., 2018a), with a one factor structure, good test-retest reliability (test-retest correlations of 0.55), and a Cronbach's Alpha coefficient of .73 at age 14 and .70 at age 16. The scale consists of 8 items tapping into self-control with items such as "I fail to finish things that I start" and "I am inattentive or easily distracted". The response format of the items is a 3-point scale, with response options *Not True* (coded 0), *Somewhat or Sometimes True* (coded1), and *Very True or Often True* (coded 2). We recoded the items such that higher sum scores reflected higher overall levels of self-control.

Statistical analyses

All analyses were conducted in M*plus* version 7 (Muthén & Muthén, 2012), evaluating goodness of fit using the Root Mean Square Error of Approximation (RMSEA) and the Comparative Fit Index (CFI) with the cut-off scores defined by Hu & Bentler (1999). In a series of bivariate twin saturated models, means and variances, by zygosity and gender, were estimated. Nested models were compared by hierarchic χ^2 tests, computing the χ^2 by subtracting -2LL (log-likelihood) for the full model from that for a reduced model (v2 = -2LL1 - (- 2LL0)). A more constrained model is kept as a better fitting model if the fit of the constrained model was not significantly worse than the fit of the more saturated model (α <.01 as level of significance). We computed phenotypic correlations while controlling for non-independence of observations by clustering data around the family identification variable, using the 'complex option' in M*plus* (Rebollo, de Moor, Dolan, & Boomsma, 2006).

Bivariate twin analyses

We applied bivariate twin analyses to investigate to what extent genetic and environmental factors contribute to family connectedness and self-control in adolescence. The observed phenotype and the association between two phenotypes can be decomposed into 1) genetic effects which can be additive genetic effects (A), and/or dominance genetic effects (D), 2) shared environmental effects (C), and 3) unique environmental effects (E), which is the part of the total variance that is unique to a certain individual, and also includes measurement error (see Figure 1A). Additionally, it allows us to calculate genetic (r_g) and environmental correlations (r_e), which quantify the extent to which an association is influenced by the same genes or by the same environmental factors (see Figure 1B).

Monozygotic-twin difference models

To further explore direction of causation between family connectedness and self-control in adolescence, we applied monozygotic-twin difference models. First, we applied *monozygotic within-pair difference* analyses. Here we calculated a within-pair difference score (score MZ twin 2 – score MZ twin 1), and assessed whether the differences between twins on one trait significantly predict differences on the other trait. Longitudinally, we assessed whether the difference in family connectedness between twins at age 14 predicted differences at age 16 in self-control, and vice versa, whether differences in self-control between twins at age 14 predicted differences in family connectedness

at age 16. Second, we applied *monozygotic within-individual change* models. This is a method to test whether the monozygotic twin who perceives an increase in family connectedness from age 14 to age 16 shows an increase in self-control from age 14 to age 16, as compared to the co-twin with a lesser increase in family connectedness over time (see Figure 2).

Because monozygotic twins are genetically identical, any phenotypic difference between them cannot be the result of genetic influences. These models are therefor particularly strong, because it examines an association controlling for genetic confounding and under the causal hypotheses these monozygotic differences should be significantly associated (Bartels et al., 2012; De Moor et al., 2008).

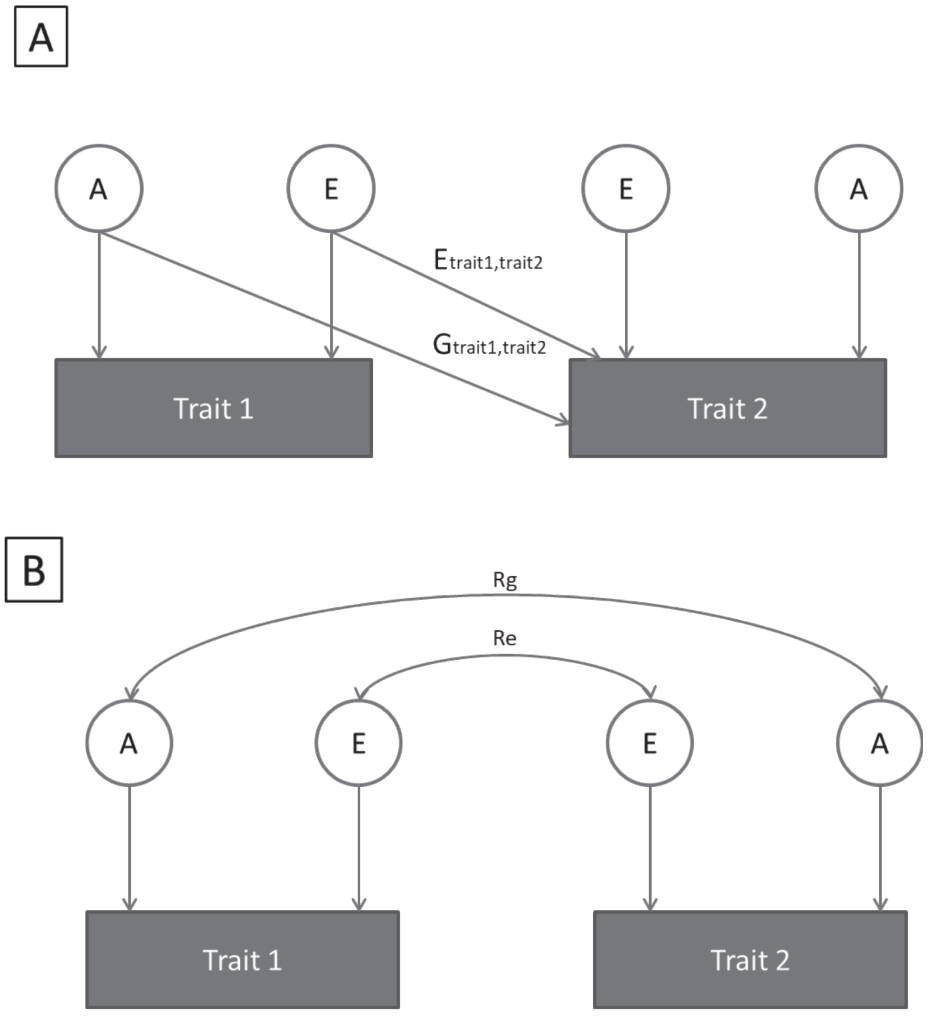

Figure 1. Graphical representation of the longitudinal bivariate twin models.

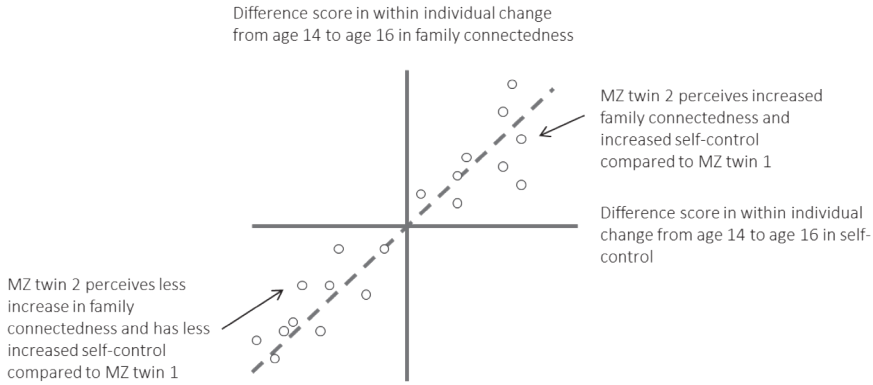

Figure 2. Graphical representation of the expected results of monozygotic within-individual change models. When the association is not explained by genetic confounding, one would expect that the twin who perceives increased family connectedness from age 14 to age 16 shows increased self-control from age 14 to age 16, as compared to his/her genetically identical co-twin

RESULTS

Descriptives

Means, standard deviations, and sample sizes are provided in Table 1. There were no significant mean or variance differences between boys and girls, nor between monozygotic and dizygotic twins. Constraining the twin correlations to be equal across gender did not deteriorate model fit, indicating the absence of gender differences in the genetic architecture of family connectedness or self-control, respectively (see Table S1, Supplemental Material).

Is there an association between family connectedness and self-control in adolescence?

The phenotypic correlations between family connectedness and self-control are presented in Table 1. All correlations were small, positive, and significant, with a cross-sectional correlation of r=.21, p<.001, 95% CI [.19, .23] at age 14, and a cross-sectional correlation of r=.19, p<.001, 95% CI [.17, .21] at age 16. The longitudinal association between family connectedness at age 14 and self-control at age 16 was r=.17, p<.001, 95% CI [.14, .20]. The longitudinal association between self-control at age 14 and family connectedness at age 16 was r=.15, p<.001, 95% CI [.12, .18].

These results suggest that family connectedness and self-control are associated in adolescence, with more family connectedness associated with higher adolescent self-control and, vice versa, higher adolescent self-control associated with more family connectedness. Thus, on average, in families where adolescents perceive more family connectedness, adolescents show higher self-control and, vice versa, adolescents with higher self-control perceive their families to be more connected, albeit with a small effect size.

Table 1. Means, standard deviations, and phenotypic correlations with 95% confidence intervals

#	Variable	Age	*M*	*SD*	*N*	1	2	3
1	Family Connectedness	14	19.42	2.78	10,685			
2	Self-Control	14	11.74	2.76	11,260	.21* [.19, .23]		
3	Family Connectedness	16	19.04	2.87	7,430	.42* [.38, .45]	.15* [.12, .18]	
4	Self-Control	16	11.64	2.69	8,175	.17* [.14, .20]	.58* [.56, .60]	.19* [.17, .21]

Note. * $p<.01$

What are the genetic and environmental contributions to this association?

Twin correlations. The twin correlations are summarized in Table 2. All correlations were significant, and all the monozygotic twin correlations were stronger than the dizygotic twin correlations, suggesting the presence of genetic influences on both family connectedness and self-control. For family connectedness, the dizygotic twin correlations were close to the monozygotic twin correlations, which suggests an influence of the shared environment (C), while for self-control the monozygotic correlations were more than twice as high as the dizygotic correlations, indicating the presence of genetic dominance (D).

Bivariate twin model. The results of the bivariate genetic twin model are presented in Table 3 (univariate standardized A, C/D, E estimates for family connectedness and self-control respectively are presented in Table S2, Supplemental Material). We modelled the bivariate genetic, and unique environmental effects but not the bivariate shared environmental effects, because it is not possible to estimate both C and D in the same model, and shared environmental influences did not contribute to variation in self-control (Willems et al. 2019b.). All bivariate twin models showed good model fit (see Table S3, Supplemental Material).

For the cross-sectional associations, genetic factors largely contributed to the association between family connectedness and self-control. At age 14, the covariation between family connectedness and self-control was for 83%, p<.001, 95% CI [.71, .94] explained by genetic factors and for 18%, p <.01, 95% CI [.06, 29] by nonshared environmental factors. At age 16, the covariation was for 66%, p<.001, 95% CI [.50, .83] explained by shared genetic factors and for 34%, p<.001, 95% CI [.17, .50] by nonshared environmental factors.

For the associations from age 14 to age 16, genetic factors solely contributed to the link between family connectedness and self-control. The association between family connectedness at age 14 and self-control at age 16 was for 95%, p<.001, 95% CI [.76, .100] explained by common genetic factors. Environmental effects on the covariance were non-significant: 5%, p=.96, 95% CI [-.23, .24]. Similarly, the association between self-control at age 14 and family connectedness at age 16 was for 72%, p<.001, 95% CI [.47, .97] explained by common genetic factors. Environmental effects on the covariance were non-significant: 28%, p=.03, 95% CI [.03, .54].

Table 2. Twin correlations and cross-twin cross-trait correlations with 95% confidence intervals

Correlations	**MZ**	**DZ**
Twin Correlations		
Family Connectedness Age 14	.35*[.31, .39]	.25*[.21, .29]
Self-Control Age 14	.49*[.46, .53]	.18*[.14, .22]
Family Connectedness Age 16	.39*[.34, .44]	.24*[.19, .29]
Self-Control Age 16	.47*[.43, .51]	.18*[.13, .23]
Cross-Twin Cross-Trait Correlations		
Family Connectedness Age 14 - Self-Control Age 14	.18*[.15, .20]	.09*[.07, .10]
Self-Control Age 16 - Family Connectedness Age 16	.12*[.09, .16]	.06*[.04, .08]
Family Connectedness Age 14 - Self-Control Age 16	.17*[.14, .21]	.09*[.06, .11]
Self-Control Age 14 - Family Connectedness Age 16	.11*[.06, .15]	.05*[.03, .08]

Note. *α<.01

While the bivariate heritability estimates elucidate the contribution of genetic and environmental factors to the phenotypic association between family connectedness and self-control, the genetic correlations (r_g) and environmental correlations (r_e) quantify the extent to which the two are influenced by the same genes or by the same environmental factors. These correlations are

presented in Table 3 and showed that r_g ranged between .36 and .63 while r_e ranged between .00 and .07. This indicates that, over and above the strong influence of genes on the association between family connectedness and self-control, there is also overlap between the genes involved in both traits. The overlap between environmental factors is small or close to zero.

Table 3. Genetic and environmental contributions (95% Confidence Interval) to the association between family connectedness and self-control

	A	E	Rg	Re
Cross-sectional				
Family Connectedness Age 14 - Self-Control Age 14	.83*[.71, .94]	.18*[.06, .29]	.63*[.40, .86]	.06*[.02, .10]
Family Connectedness Age 16 - Self-Control Age 16	.66*[.50, .83]	.34*[.17, .50]	.37*[.21, .52]	.10*[.05, .15]
Longitudinal				
Family Connectedness Age 14 - Self-Control Age 16	.95*[.76, 1.00]	.05 [-.23, .24]	.57*[.34, .81]	.00 [-.07, .07]
Self-Control Age 14 - Family Connectedness Age 16	.72*[.47, .97]	.28 [.03, .54]	.36*[.20, .57]	.07 [.01, .14]

Note. A= genetic contributions to the overlap between family connectedness and self-control, E= the environmental contribution to the overlap between family connectedness and self-control, Rg = genetic correlations, Re = environmental correlation. *α <.01

Is there a direction of effect between family connectedness and self-control?

Monozygotic twin within-pair differences model. The monozygotic twin within-pair difference between family connectedness at age 14 and self-control at age 16 was not significant: r=.00, p=.99, 95% CI [-.09, .09]. Similarly, the monozygotic twin within-pair difference between self-control at age 14 and family connectedness at age 16 was not significant: r=.07, p=.13, 95% CI [-.02, .16].

Monozygotic twin within-individual change model. The results show that, in genetically identical twin pairs, the twin showing the largest increase in family connectedness from age 14 to age 16 did not report larger increase in self-control from age 14 to age 16 than the co-twin showing lower increase (or even decrease) in experienced family connectedness: r=.01, p=.83, 95% CI [-.08, .10].

DISCUSSION

The present study investigated the association between family connectedness and self-control, examining whether the association still holds in adolescence as well as the nature of this association. In line with the literature on early and middle childhood, the results confirmed that more perceived family connectedness is related to better self-control in adolescents, albeit with a small effect size. When investigating the nature of this association, we found that this correlation was mainly explained by common genetic factors, with the effects of environmental factors being small. That is, the monozygotic twin who perceives more family connectedness did not show higher self-control than his/her co-twin and, vice versa, the monozygotic twin with more self-control did not perceive more family connectedness than his/her co-twin.

These findings suggest that while the association between family connectedness and self-control holds in adolescence, the two traits are not likely to causally influence one-another over time. Rather, an underlying common factor such as genetic pleiotropy with some additional unique environmental influences, seems to drive their association. Although there is common awareness that correlation does not equal causation, past research may have overestimated the association and too quickly concluded that the significant phenotypic relationship implies a transfer effect. For example, earlier studies investigating person-environment interactions emphasise how socializing processes are the driving source behind optimal self-control development (Craig, 2016; Moilanen, Rasmussen, & Padilla-Walker, 2015; Janssen et al., 2016). The results of this study emphasize that understanding person-environment transactions are more complex, specifically highlighting the key role of biological factors in socializing processes. As such, if we aim to understand the mechanisms underlying person-environment transaction, it is important to incorporate both environmental *and* biological factors (Mõttus et al., 2019). Combining these factors, thereby bridging multiple scientific disciplines, is necessary to paint a more complete picture of the aetiology of individual differences in self-control.

An explanation for the role of shared genetic factors is that individuals evoke an environment as a result of their person characteristic: their self-control evokes more family connectedness and this is genetically mediated (evocative gene-environment correlation, Plomin & Daniels, 2011). Passive gene-environment correlation could also be a possible explanation, when there is a correlation between the environment the child is raised and the genotype

a child inherits. For example, parents with high self-control are more likely to create a house environment with family connectedness but also transfer their 'self-control' genes to their children (Plomin & Daniels, 2011). Perhaps gene environment interaction is also at play, with certain genotypes being more sensitive to certain environments, positive or negative, as suggested by the diathesis stress model and the differential-susceptibility theory (Belsky, Bakermans-Kranenburg & van Ijzendoorn, 2007; Monroe & Simons, 1991).

A limitation of the present study is that while we can investigate the nature of the association, we cannot unravel the more complex mechanisms underlying the association. As such, we do not know whether the association is explained by genetic pleiotropy, gene-environment correlation, gene-environment interaction, or a combination of these. Research designs such as children of twin designs, sibling designs, adoption studies, transmitted versus non-transmitted alleles, and interactions between polygenic risk scores and environments would allow for a deeper understanding of such mechanisms and are highly recommended for future research (Connolly, 2019; Kong et al., 2018; Leve et al., 2013; Malanchini et al., 2017; McAdams et al., 2018; Peyrot et al., 2014; Willems et al., 2019b). Another limitation of the study is that we focused a certain period in adolescence (i.e., adolescents aged 14 and 16). Whereas the current approach allowed to examine cross-age effects of family connectedness on self-control and vice versa, longitudinal studies stretching across childhood would be an important next step in our understanding of the way adolescents develop within, and in interaction with, their social world. It is also important to emphasize that we only included self-reports, possibly inflating person-specific covariances due to single-informant non-random error, and we recommend replication of our findings in the future applying a multiple-rater approach.

As a concluding note, it is important to emphasize that twin models assess variance differences not mean change (Harold, Leve & Sellers, 2017). So, while we do see that change occurs over time – family connectedness and self-control are significantly and positively associated over time – the current results demonstrate that most variance is explained by family members sharing the same genes. This implies that while we can still apply association studies such as correlational analyses and cross-lagged panel models, we should be more careful in the interpretation of the underlying causes of such associations. That is, phenomena we see within families can also be the product of parent and children sharing the same genes rather than being exclusively attributable to environmental processes.

SUPPLEMENTS

CHAPTER 6

Table S1. Model fitting assessing means and variance differences for family connectedness and self-control age 14 and age 16, respectively

#	Model	EP	-2LL	Comp.	χ2	Δdf	*p* value
Age 14							
0	Saturated	70	-43570.55	-	-	-	-
1	Birth Order - Equal Means and Equal Variances	54	-43575.19	0	4.64	16	1.00
2	Zygosity - Equal Means	46	-43582.87	1	7.68	8	0.96
3	Zygosity - Equal Variances	38	-43587.38	2	4.51	8	0.81
4	Gender - Equal Means	36	-43590.74	3	3.36	2	0.19
5	Gender - Equal Variances	34	-43592.79	4	2.05	2	0.36
6	MZm = Mzf & DZm = DZf	22	-43598.54	5	5.76	12	0.93
7	DZ = Dos	16	-43601.91	6	3.36	6	0.76
Age 16							
0	Saturated	70	-30150.85	-	-	-	-
1	Birth Order - Equal Means and Equal Variances	54	-30157.68	0	6.83	16	0.98
2	Zygosity - Equal Means	46	-30173.11	1	15.43	8	0.05
3	Zygosity - Equal Variances	38	-30177.51	2	4.40	8	0.82
4	Gender - Equal Means	36	-30182.00	3	4.49	2	0.81
5	Gender - Equal Variances	34	-30186.61	4	4.61	2	0.10
6	MZm = Mzf & DZm = DZf	22	-30196.62	5	10.01	12	0.62
7	DZ = Dos	16	-30198.25	6	1.63	6	0.95

Note. ep = estimated parameters, -2ll = minus 2 loglikelihood, Comp= model compared to, χ2 = chi square, df = degrees of freedom, MZm= monozygotic male twins, MZf= monozygotic female twins, DZm=dizygotic male twins, DZf=dizygotic female twins, Dos = dizygotic opposite gender twins. In model 6, the correlations between monozygotic males and monozygotic females, and the correlations between dizygotic males and females were constrained to be equal. In model 7, the correlations between dizygotic same-sex and opposite-sex were constrained to be equal.

Table S2. Additive genetic (A), dominant genetic (D), common shared environmental (C), and unique environmental (E) estimates [95% Confidence Interval] to family connectedness and self-control, respectively

Age	Family connectedness			Self-control		
	A	C	E	A	D	E
Age 14	.19 [.08, .31]	.15 [.07, .24]	.65 [.61, .70]	.22 [.08, .37]	.27 [.11, .43]	.51 [.47, .54]
Age 16	.30 [.16, .45]	.09 [-.02, .20]	.61 [.56, .66]	.23 [.04, .43]	.23 [.03, .44]	.53 [.49, .58]

Table S3. Estimated parameters (EP) and model fit of bivariate twin models

Model	EP	RMSEA	CFI
Bivariate twin model age 14	12	0.02	0.99
Bivariate twin model age 16	12	0.02	0.98
Bivariate twin model family connectedness age 14, self-control age 16	12	0.00	1.00
Bivariate twin model self-control age 14, family connectedness age 16	12	0.00	1.00

Note. RMSEA= Root Mean Square Error of Approximation (RMSEA), CFI= Comparative Fit Index

Chapter 7

Out of control: Examining the association between family conflict and self-control in adolescence in a genetically sensitive design

ABSTRACT

Objective: Family conflict is associated with low self-control in adolescence. Thus far research about the direction of this association is inconclusive. In this study, we sort out whether this association reflects a causal effect or whether it is explained by a common underlying cause, including genetic factors. **Method:** In twin data, we fitted a series of causal models, and compared models for the association of family conflict and self-control including reciprocal causation, unidirectional causation from family conflict to low self-control, unidirectional causation from low self-control to family conflict, and common genetic susceptibility. We included data of a large sample of twins aged 14 (*N*=9,173), all enrolled in the Netherlands Twin Register. **Results**: The results suggested a unidirectional pathway model where family conflict leads to low self-control in adolescence, with genetic factors also playing a role in explaining the association. **Conclusion:** Adolescents experiencing family conflict are at risk to show hampered self-control capacities, with family conflict being a robust predictor of low self-control through common genetic factors but also through direct causal influences.

Keywords: family conflict; self-control; adolescence; twins; environment; genetics

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Adolescence is marked by a range of self-control challenges. Adolescents have to finish their homework while tempted to check social media feeds, conform to parental rules while striving for independence, and regulate insecurities when exposed to picture perfect Instagram posts. Not being able to exert self-control – the inability to alter unwanted impulses and behaviour, in order to bring them into agreement with goal-driven responses – places adolescents at risk for myriad negative outcomes (Bridgett, Burt, Edwards, & Deater-Deckard, 2015; Nigg, 2017). Especially during adolescence, characterized by a range of normative biological and social changes, self-control is key to a successful transition into adulthood (Crone & Dahl, 2012). For example, youth who exhibit low self-control are more likely to fail in school, drink alcohol, be arrested for crimes, and develop psychiatric disorders (Duckworth & Seligman, 2012; Finkenauer, Engels, & Baumeister, 2005; Vazsonyi, Mikuska, & Kelley, 2017). Because low self-control is a powerful predictor of health, wealth, and public safety across the lifespan (Caspi et al., 2016; Moffitt et al., 2011), it is important to identify factors shaping its development, including contextual factors such as family conflict (Finkenauer, Buyukcan-Tetik, Schoemaker, Willems, Bartels, & Baumeister, 2018; Finkenauer et al., 2015).

Growing evidence indicates that family conflict – relational escalations where one or more family members engage in physical and verbal aggression – is associated with self-control problems. However, adolescents are not passive recipients to their environment and the way family conflict and low self-control are associated is complex. While some association studies find that family conflict predicts low self-control (Schwarz, Stutz, & Ledermann, 2012), others find that adolescents' low self-control predicts family conflict (Brody & Ge, 2001), and again others suggest a reciprocal relationship (Hallquist, Hipwell, & Stepp, 2015; Tiberio et al., 2016).

How are family conflict and low self-control associated?

Relational escalations and the coinciding unsafe and unpredictable family environment can undermine children's ability to regulate and alter undesirable impulses, behaviors, and emotions (Bridgett et al., 2015; Davies & Cummings, 1994; Finkenauer et al., 2015). Findings from longitudinal studies demonstrate that children exposed to chronic, hostile, or poorly resolved family conflicts exhibit lower self-control (Davies, Cicchetti, & Martin, 2012; Sturge-Apple, Davies, Cicchetti, Hentges, & Coe, 2016). In addition, family conflict may have an indirect effect on children's self-control, mediated through other family processes such as poor parenting practices (Krishnakumar & Buehler, 2000),

insecure parent-child relationships (Davies & Cummings, 1994), and chaotic household conditions (Whitesell, Teti, Crosby, & Kim, 2015).

Alternatively, evidence suggests that low self-control predicts conflict. Individuals with low self-control are more likely to behave more aggressively towards strangers (DeWall, Baumeister, Stillman, & Gailliot, 2007), and their romantic partner (Finkel, DeWall, Slotter, Oaten, & Foshee, 2009). Individuals with low self-control trigger distrust within relationships and are less successful in de-escalating conflict (Righetti & Finkenauer, 2011). Also, low self-control is a modest yet consistent predictor of victimization, suggesting that low self-control may evoke aggression in others (Pratt, Turanovic, Fox, & Wright, 2014).

Longitudinal studies investigating a reciprocal association between family conflict and self-control in adolescence are scarce and yield inconsistent results (Schwarz et al., 2012). One study tracked the development of family conflict and low self-control across early adolescence (from age 12 to age 13, N = 120, Brody & Ge, 2001). They found that low self-control was linked to conflict one year later, but conflict was not related to lower self-control one year later. Another study assessed family conflict and low self-control repeatedly over five years from middle childhood into adolescence (N = 2450, Hallquist et al., 2015). They found reciprocal effects, with earlier poor self-control predicting later conflict and earlier conflict predicting later poor self-control. In contrast, another study following adolescents from age 11 to age 16 (N = 473), illustrated no significant bidirectional effects between family conflict (e.g., verbal hostility and punitive communication) and low self-control (Moilanen, Rasmussen, & Padilla-Walker, 2015). This indicates uncertainty still exists about the direction of causation between family conflict and low self-control.

Genetic contributions to family conflict and self-control

Importantly, caution is warranted when interpreting these effects because the findings are likely to be confounded by genetic factors that influence both exposure and outcome (D'Onofrio, Lahey, Turkheimer, & Lichtenstein, 2013). Adolescent twin studies show that individual differences in family conflict and self-control, respectively, are partly influenced by genetic factors. Heritability estimates range between 18% and 31% for family conflict based on adolescent self-report and between 44% and 64% for self-control based on adolescent self-report (Beaver, Ratchford, & Ferguson, 2009; van der Aa, Boomsma, Rebollo-Mesa, Hudziak, & Bartels, 2010; Willems et al., 2018a). Given the known genetic contributions to both, it may thus well be that observed associations are explained by common genetic factors that simultaneously

influence both family conflict and low self-control rather than by a direct relation.

Current study

A design taking into account genetic and environmental sources of variance while simultaneously modelling the direction of effect is the 'direction of causality' model (DoC model, Duffy & Martin, 1994; Heath et al., 1993). The DoC model predicts different cross-sectional cross-twin cross-trait correlations (i.e., the correlation between family conflict in twins with self-control in his/her co-twin) depending on differences in heritability between two traits, allowing to make a prediction considering the direction of the effect. This model has been applied successfully to address directionality in earlier studies (Gillespie, Zhu, Neale, Heath, & Martin, 2003; van Bergen et al., 2018). Thereby, this method allows us to statistically test whether the cross-twin cross-trait correlations reflect (1) a unidirectional effect where family conflict predicts low self-control, (2) a unidirectional effect where low self-control predicts family conflict, (3) a reciprocal effect, where family conflict and low self-control influence each other bidirectionally, or (4) a common genetic factor driving the association between family conflict and low self-control. In the present study, we apply the direction of causality model to elucidate the relationship between family conflict and self-control in a large sample of adolescent twins.

METHOD

Sample and procedure

The Netherlands Twin Register (NTR) is a population-based study initiated in 1987 in the Netherlands, following twins and their families from birth till adulthood with age-specific assessments. In the current study we include data of 14-year-old twins who, upon parental consent, received questionnaires with questions on physical and psychological well-being and family functioning (van Beijsterveldt et al., 2013). Data collection was approved by the Medical Ethical Committee at the VU medical center (2003/182).

The sample consisted of 9,173 14-year-old twins (M_{age}=14.66, SD_{age}=0.64, 57.6% female participants, complete twin pairs: 85%, monozygotic twin pairs (MZ), n=1,861, dizygotic twin pairs (DZ), n=3,315). Participants came from all regions of the Netherlands, both rural and urban areas, and were primarily Caucasian. For 28.1% of the same-sex twin pairs, zygosity was

determined based on DNA typing or blood group. For the remaining same-sex pairs, zygosity was determined based on items concerning physical similarities rated by their parents. Earlier research showed these items allow for accurate determination of zygosity in 93% of the cases (Rietveld et al., 2000).

Measures

Family conflict. This study used the Dutch translation of the Conflict subscale from the Family Environment Scale (FES) to assess adolescents' perception of family conflict (De Coole & Jansma, 1983). This subscale consists of 11 items, measuring the amount of conflict, aggression, and openly expressed anger within the family, yielding a Cronbach's alpha coefficient of 0.72. For example, the scale included statements such as "we argue a lot at home" and "sometimes family members get so angry, they throw things". Participants were asked to indicate if these statements were applicable to their family (1 = *No,* 2 = *Yes*), with higher scores indicating more conflict.

Self-control. We used the eight item ASEBA Self-Control Scale (ASCS) to assess self-control of adolescents (Willems et al., 2018a). The ASCS is a psychometrically sound construct, with solid construct validity (one factor structure), acceptable internal consistency (Cronbach's alpha coefficient of 0.73), and good test-retest reliability (test-retest correlation of 0.55, Willems et al., 2018a). The scale consists of items of the aggression problem scale, attention problem scale, and rule breaking scale of the ASEBA such as "I fail to finish things that I start" and "I am inattentive or easily distracted"(Achenbach & Rescorla, 2001). The response format of the items is a 3-point scale, with response options *Not true* (coded 0), *Somewhat or Sometimes True* (coded 1), and *Very True or Often True* (coded 2). An overall score for aggression, attention, and rule breaking problems, respectively, was created. These score taps into self-control problems, with higher scores reflecting lower overall levels of self-control. From earlier research, we know that the combination of genetic effects and unique environmental effects on the ASCS is similar to the estimates of other aspects of self-regulation such as effortful control, impulsivity, and attentional control (Fagnani et al., 2017).

Statistical analyses

The association between family conflict and low self-control was tested in three consecutive steps, each model that was tested formed the basis for the next step, and all analyses were performed in M*plus* (Muthén & Muthén, 2012). We applied robust maximum likelihood (MLR) as an estimator, providing less-

biased standard errors (Maydeu-Olivares, 2017). The model's M*plus* syntax is provided in Supplement 4, available online.

1. Measurement model

While the measurement model of the ASCS has been studied previously(Willems et al., 2018a), the factor structure of the family conflict scale has not yet been tested in our sample. Earlier studies reported a unidimensional structure of family conflict (De Coole & Jansma, 1983), Accordingly, we conducted a confirmatory factor analysis in our data confirming the unidimensional structure. Next, we applied a correlational model, including measurement models for both traits, to assess the phenotypic correlation between family conflict and low self-control. In order to correct for the dependency of the observations due to clustering in families, we applied a sandwich estimator (Rebollo, de Moor, Dolan, & Boomsma, 2006).

2. Direction of causality

The model including both measurement models formed the basis for the subsequent analyses: assessing the direction of causation (DoC, Duffy & Martin, 1994; Heath et al., 1993). In a series of saturated models, we tested equality of means and variances across zygosity and gender. The DoC design is built on the classical twin method with the premise that differences in the resemblance between monozygotic twins (sharing approximately 100% of their segregating genes) and dizygotic twins (sharing 50% of their segregating genes on average) can be used to parse phenotypic trait variance into genetic and environmental variance. If monozygotic twins are more alike than dizygotic twins, genetic influences are indicated. Often, the total variance of a trait and the covariance between traits is decomposed into additive genetic (A, additive effects of alleles at multiple loci), dominance genetic (D), or common environment (C, the part of the variance that is shared by members of family), and non-shared environment (E, the part of the total variance that is unique to a certain individual) variances. Residual (co)variances of the items were also decomposed into genetic and environmental effects.

Based on the literature, we know that differences in family conflict are mainly due to differences in environment (van der Aa et al., 2010). This is reflected in the twin correlations of family conflict, with the DZ correlation being close to the MZ correlation. On the contrary, self-control is more heritable than family conflict, with the MZ correlation approximately twice as high as the DZ correlation (Willems et al., 2018a). This difference in the pattern of

the MZ and DZ correlation is utilized by the DoC model to make a prediction regarding the direction of the effect. Instead of focusing on the univariate twin correlations, the DoC model examines the cross-twin cross-trait correlations (i.e., the correlation between family conflict in twins with self-control in his/her co-twin), and tests specific predictions regarding the pattern of the MZ and DZ cross-twin cross-trait correlations.

If family conflict (low heritability) unidirectionally predicts self-control (high heritability), the cross-twin cross-trait correlations should reflect a DZ correlation that is close to the MZ correlation, mirroring the genetic architecture of family conflict. If self-control unidirectionally predicts family conflict, the cross-twin cross-trait correlations should reflect MZ correlations that are approximately twice as high as the DZ correlations, mirroring the genetic architecture of self-control. If the association is bidirectional, the cross-twin cross-trait correlations reflect a combination of the MZ and DZ pattern of family conflict and self-control (Heath et al., 1993). Structural equation modelling allows us to assess which of these directional models fit the cross-trait cross-twin correlations best.

3. Bivariate genetic correlational model

Considering the heritability of family conflict and low self-control, their association might be explained by a common underlying genetic factor instead of a causal effect. Therefore, we also applied a bivariate genetic correlational model to investigate the relative contributions of genetic and environmental factors to the variance in family conflict and self-control and their covariance. We opted for adding a genetic correlation (denoted as Rg), rather than a non-shared correlation (denoted as Re) because of the major contribution of genetics on individual differences in self-control (Willems et al., 2018a).

Assessing model fit

Several indices were applied to assess which of the aforementioned models was most likely to be reflected by the data. Goodness of fit was evaluated using the Root Mean Square Error of Approximation (RMSEA) and the Comparative Fit Index (CFI), adopting the cut-off scores defined by (Hu & Bentler, 1999). Nested submodels (e.g., bidirectional vs. unidirectional models) were compared by hierarchic χ^2 tests. The χ^2 statistic was computed by subtracting—2LL (log-likelihood) for the full model from that for a reduced model (v2=−2LL1−(−2LL0)). If a *p*-value higher than 0.01 was obtained from the χ^2-test, the fit of the constrained model was not significantly worse than the fit of the

more complex model. In this case, the constrained model was kept as the most parsimonious and best fitting model. To compare non-nested models (e.g., direction of causation models vs. bivariate genetic correlational model), we applied Akaike's Information Criteria (AIC). The AIC addresses the trade-off between descriptive accuracy and parsimony of the model, with lower AIC indicating a better fit of the model to the observed data. To better understand AICs of competing models, we computed AIC weights (in R, with the Multi-Model Inference "MuMIn" Package, Barton, 2018). AIC weights are ratios that reflect differences in AIC with respect to the AIC of the best candidate model, thereby obtaining estimates of the relative likelihood of the model (Wagenmakers & Farrell, 2004). The convenience of AIC weights is that they are distributed according to relative probability, translated to percentages, so they have interpretable meaning: ranging between 0% = very unlikely to 100% = very likely that the model represents the true model. This allowed us to quantify the amount of statistical confidence for each of the models, providing insights into the relative advantage of competing models (Wagenmakers & Farrell, 2004).

RESULTS

Descriptive statistics. There were no significant mean or variance differences in family conflict and self-control between monozygotic and dizygotic twins, nor between boys and girls. On average, adolescents scored *M*=14.34 (*min*=11, *max*=22, *SD*=2.45) on family conflict, and *M*=4.23 (*min*=0, *max*=16, *SD*=2.76) on self-control. All MZ correlations were higher than DZ correlations, suggesting a role of genetic effects for both family conflict and self-control (see Table 1). For family conflict, DZ correlations were close to MZ correlations, implying a role of the shared environment. For self-control, MZ correlations were twice as high as DZ correlations implying a role of dominant genetic influences.

Table 1. Twin Correlations and Cross-Twin Cross-Trait Correlations

	MZ	DZ
Twin Correlations		
Family Conflict	0.73 [0.69, 0.77]	0.62 [0.58, 0.66]
Low Self-control	0.60 [0.58, 0.63]	0.32 [0.25, 0.38]
Cross-Twin Cross Trait-Correlations		
Family Conflict – Low Self-control	0.46 [0.41, 0.50]	0.33 [0.29, 0.37]

Measurement model. Applying a 1-factor model for the family conflict scale suggested room for improvement (RMSEA =.038 ; CFI =.930 ; EP=33). Upon inspection of the modification indices, we allowed the residuals of items "hitting" and "swearing" to correlate. The correlation between these items suggests that both tap into more expressive forms of family conflict. This revised model fit significantly better than the initial model $\Delta\chi^2(df=1) = 109.94$, $p < .001$, and showed good overall model fit for the 1-factor model (RMSEA =.031; CFI =.95; EP=34; see Supplement 1 available online). The 1-factor structure of the self-control scale has been tested elaborately in our data published in earlier work (Willems et al., 2018a), based on sum scores of 'attention problems', 'aggression problems' and 'rule breaking' items, showing good fit (RMSEA =.00; CFI =1.00; EP=9; see Supplement 2 available online). For the phenotypic association between family conflict and low self-control, we extended the measurement models by correlating family conflict and low self-control (see Figure 1), showing good overall model fit and a moderate to strong correlation (RMSEA = .028 ; CFI =.96; EP= 44; r=.61; 95% CI .58 - .64).

Direction of causation model. The direction of causation model (DoC) works well when the phenotypic correlation between traits is robust, the traits differ in their heritability, and measurement error is accounted for with a measurement model (Duffy & Martin, 1994; Heath et al., 1993). Considering the large phenotypic correlation between family conflict and self-control, the higher heritability of self-control (50% - 60%, Willems et al., 2018a) as compared to family conflict (30% - 40%)(van der Aa et al., 2010), the application of measurement models, and the large sample size (>9,000 twins), we were confident for the model to work well.

We decomposed the phenotypic twin correlations into the A, C (for family conflict) or D (for self-control), and E variance components. It is not possible to estimate both C and D in the same model. Based on the previous literature, and on the twin correlations, we therefore estimated an ACE model for family

conflict and an ADE model for self-control. Considering that previous work on the same data found no sex differences in heritability for family conflict nor self-control, we did not consider the genetic and environmental components to differ between boys and girls (Boisvert, Wright, Knopik, & Vaske, 2013; van der Aa et al., 2010). In line with earlier studies applying direction of causality models (Gillespie et al., 2003; van Bergen et al., 2018), residual variances and correlating residuals (for the family conflict scale) of the measurement model were also decomposed into genetic and environmental variance (see Supplement 3 available online, for the estimates). The contribution of D to low self-control was not significant. Therefore, we omitted this path, resulting in a constrained, and parsimonious model that was not significantly worse than the fit of the more complex model $\Delta\chi^2$ (*df*=1, *N*=5,176 pairs) = 2.50, p = .11. This bidirectional direction of causation model showed good model fit (RMSEA = .02 ; CFI =.95)(see Figure 2A.).

Next, we compared the unidirectional model low self-control *to* family conflict (Figure 2B) to the bidirectional model (Figure 2A), resulting in a large deterioration in fit $\Delta\chi^2$ (*df*=1, *N*=5,176 pairs)= 33.23 p < .001). This indicates that the unidirectional model from low self-control *to* family conflict shows a worse fit to the data than the bidirectional model. Alternatively, we compared the unidirectional model family conflict *to* low self-control (Figure 2C) to the bidirectional model (Figure 2A). Results showed that the unidirectional model was not significantly worse than the bidirectional model $\Delta\chi^2$ (df=1, *N*=5,176 pairs) = 2.63, p = .10. This indicates that an unidirectional model from family conflict *to* low self-control shows a better fit to the data than the bidirectional model.

Bivariate genetic correlational model. Fitting the bivariate genetic correlational model (Figure 2D) resulted in an AIC increase of 24.42 compared to the unidirectional model from family conflict *to* low self-control (Figure 2C), indicating that the unidirectional causal model fits the data better than the genetic correlational model.

Considering the high genetic correlation between the two traits (see Figure 2D), we subsequently tested the best fitting model of the direction of causation tests (unidirectional model from family conflict to self-control), and added a common genetic correlation (see Figure 3). The AIC of the model family conflict *to* low self-control including the genetic correlation (AIC=158065.41) was lower than the model family conflict *to* low self-control excluding the genetic correlation (AIC=158070.565), indicating that adding a genetic correlation fits the data well.

While it is current practice to accept a single model based on the lowest AIC value, differences in models based on AIC values are difficult to unambiguously interpret. Calculating AIC weights allow for a more straightforward interpretation (see Table 2, Wagenmakers & Farrell, 2004). The AIC weights showed that indeed the statistical confidence for the last model was stronger (Figure 3, probability of 68%) than the statistical confidence for the bidirectional model (Figure 2A, probability of 26%). Thus, the unidirectional model from family conflict to self-control (.46, 95% CI .34 - .57), including common genetic influences (R_g =.56, 95% CI .12 - .99), is most likely to be supported by the data.

Table 2. Akaike's Information Criterion (AIC) Weights of the Competing Models

Model	EP	AIC	AIC weights
Correlational	73	158094.98	0.00
Bidirectional	73	158067.31	0.26
Low self-control --> family conflict	72	158131.77	0.00
Family conflict --> low self-control	72	158070.57	0.05
Family conflict --> low self-control (with Rg)	73	158065.41	0.68

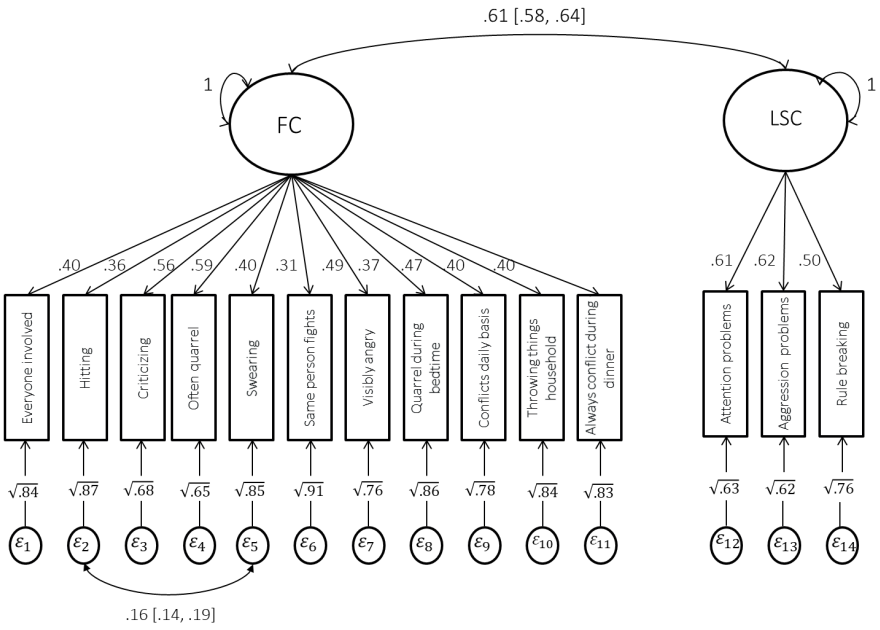


Figure 1. Measurement model, FC= family conflict, LSC= low self-control

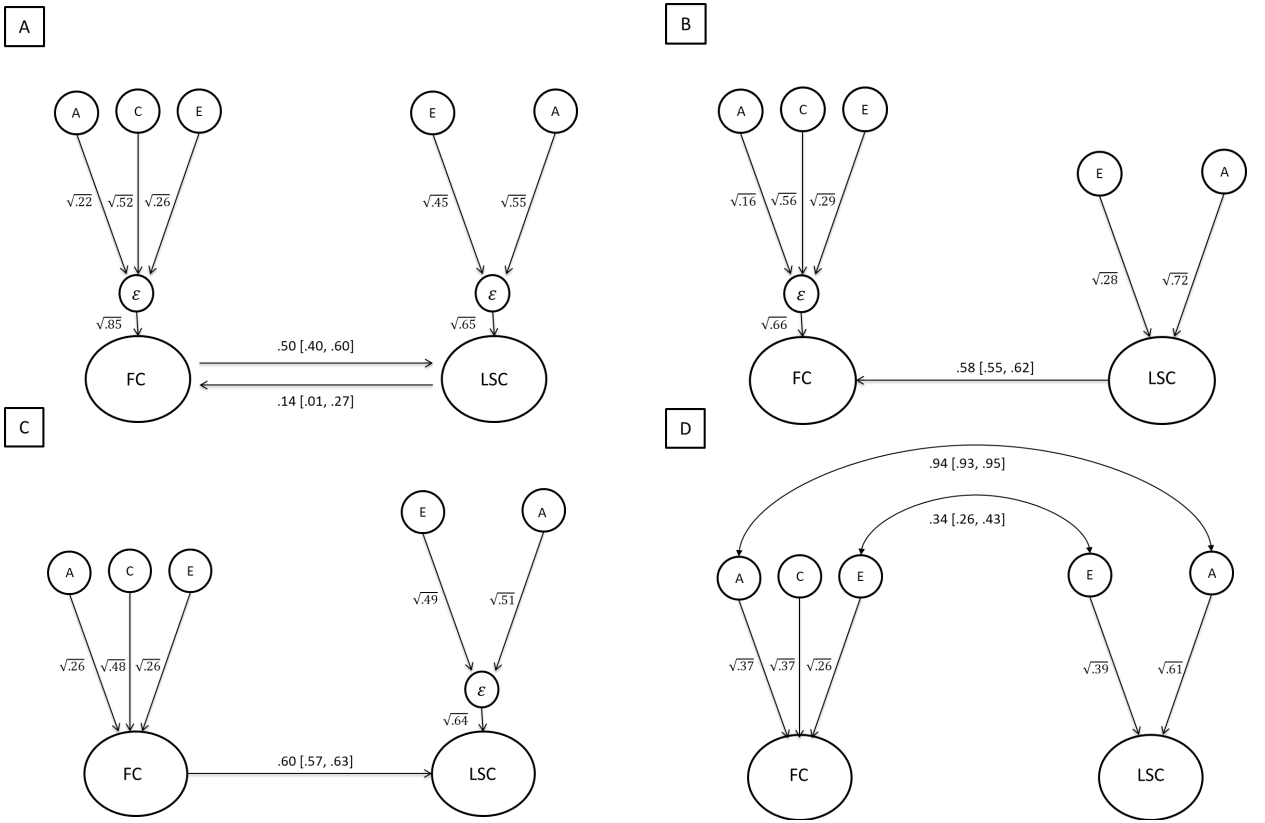


Figure 2. Direction of causality. Note: FC=family conflict, LSC= low self-control, A) bidirectional model, B) unidirectional model low self-control to family conflict, C) unidirectional model family conflict to low self-control D) bivariate genetic correlational model. In the interest of space, only the top parts of the models are shown.

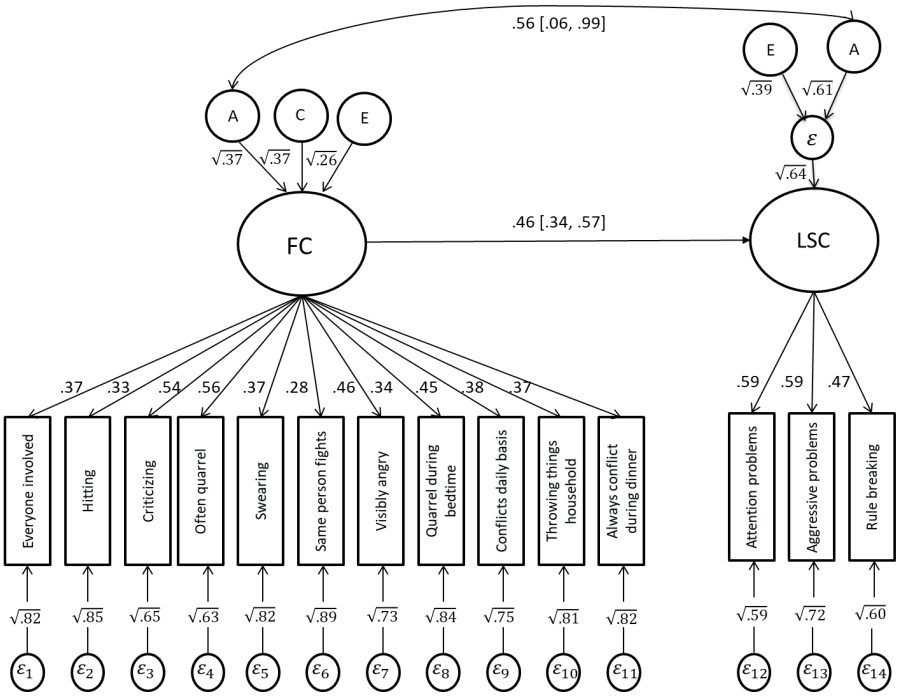


Figure 3. Unidirectional model with genetic correlation. *Note:* FC= family conflict, LSC= low self-control. In the interest of space, decomposition of residual errors, and correlation between residuals, into genetic and environmental variance is not illustrated in this figure, see Table S1. in the supplement, available online, for the estimated parameters. AIC weights show this model supports the data best

DISCUSSION

Studies consistently find that ill decisions made during adolescence due to insufficient self-control ensnare adolescents in lifestyles that have lasting effects into adulthood (e.g., poor physical health, overweight, financial issues, and substance use, Caspi et al., 2016; Moffitt et al., 2011). Supporting theoretical models (Finkenauer et al., 2018; Finkenauer et al., 2015), we found that family conflict predicts low self-control in adolescence. More specifically, in line with the self-control strength model of family violence (Finkenauer et al., 2018; Finkenauer et al., 2015), we see impairments of self-control in the wake of family conflict. To our knowledge, this is the first study to investigate this association in a large genetically sensitive design (> 9,000 twins) allowing

us to reveal the direction of effect while controlling for genetic confounds. Our results suggest a unidirectional path where family conflict predicts lower self-control in adolescence, with genetic factors also playing a role in explaining the association.

This finding indicates that families are at risk because they share the same genes, with the same genes influencing the presence of family conflict and the risk for having low self-control. Crucially, the findings suggest a directional effect of family conflict on self-control.The exertion of self-controlled behavior requires energy and resources (Finkel et al., 2009). It is possible that family conflict diminishes individual's limited resources, because it requires attention and vigilance, for example, which cannot be invested in engaging in self-control and resisting temptations (Finkel et al., 2009). Alternatively, the presence of conflict and subsequent emotional activation may impair prefrontal cortex functioning, decreasing the ability to engage self-regulatory processes (Maier, Makwana, & Hare, 2015). It is also possible that conflict gets in the way of social support and guidance necessary to develop and strengthen self-control (Krishnakumar & Buehler, 2000). And there may be other reasons, such as that family conflict possibly creates unpredictability that makes the exertion of self-control and delay of gratification disadvantageous or risky for the individual (Ellis, Bianchi, Griskevicius, & Frankenhuis, 2017).

One possible implication of our results is that practitioners and professionals should be aware that low self-control may result from the experience of conflict in the home environment rather than treating them as separate problems. Additionally, both of the underlying pathways explaining the association between family conflict and self-control – the contextual risk of family conflict and the genetic similarities within the family – are manifested at the family level. This suggests that family-based approaches for intervention or prevention strategies could be promising (Harold, Leve, & Sellers, 2017). Such approaches, targeting family conflict, might break the potentially vicious cycle of maladaptive self-control development. Empirical research with controlled trials would be needed to confirm this suggestion. Importantly, we need to acknowledge the complexity of family conflict and the environmental factors associated with it, and solely targeting family conflict is unlikely to be the one and only way to help those families at risk.

There are some limitations in this study. Establishing causality is a complex endeavor. While our results suggest causality, we cannot infer it with full certainty. Despite its powerful design, our study is based on cross-sectional data and does not explicitly model person-environment transactions

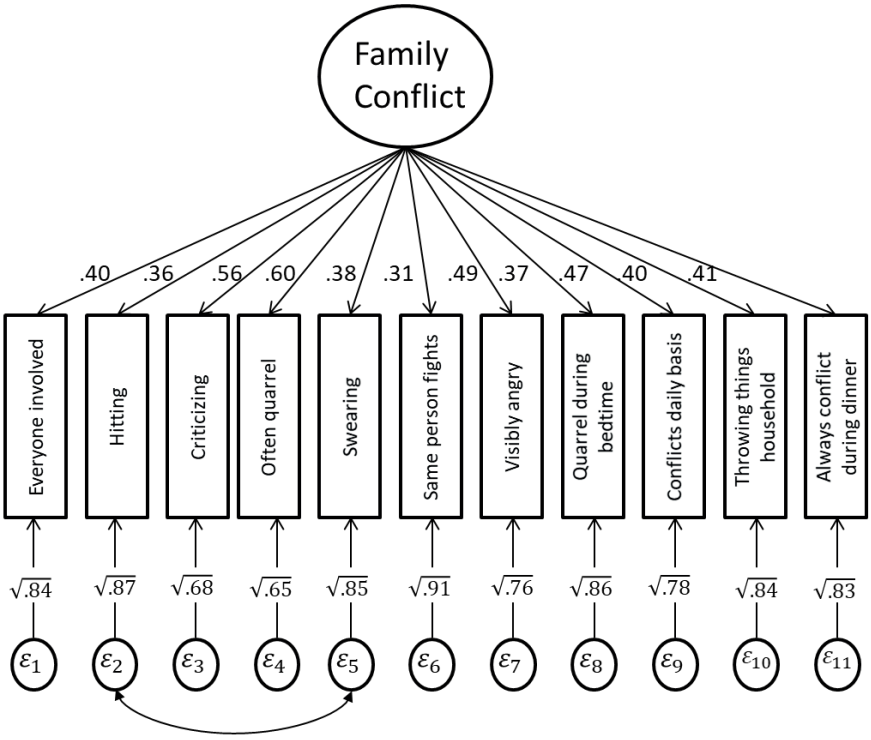
from earlier time points. Future research is needed to apply complementary research designs with longitudinal data (to see how childhood experiences shape adolescence) or observational designs (to further examine underlying mechanisms). Additionally, both family conflict and low self-control measures relied on self-reports. An important extension of our study would therefore be to include a multiple-rater approach. However, one advantage of using adolescents' self-reports is that they reflect their subjective experience, and the way their psychological reality influences their behavior may be at least as important as parental perceptions of family functioning (Hannigan, McAdams, Plomin, & Eley, 2016).

Moreover, our results show a common genetic pathway between family conflict and self-control, potentially indicating the presence of gene-environment correlation (rGE, when there is a correlation between the genotype the adolescent inherits and the environment the adolescent is raised). Unfortunately, our model does not allow to distill whether the genetic pathway reflects genetic pleiotropy or, if present, which *specific* gene-environment correlation (e.g. passive, evocative, or active gene-environment correlation, Harold et al., 2017). Future research applying adoption data or children of twin data is strongly recommended, as this allows to further unravel the dynamic processes underlying the family conflict – self-control link (Leve et al., 2013; McAdams et al., 2018).

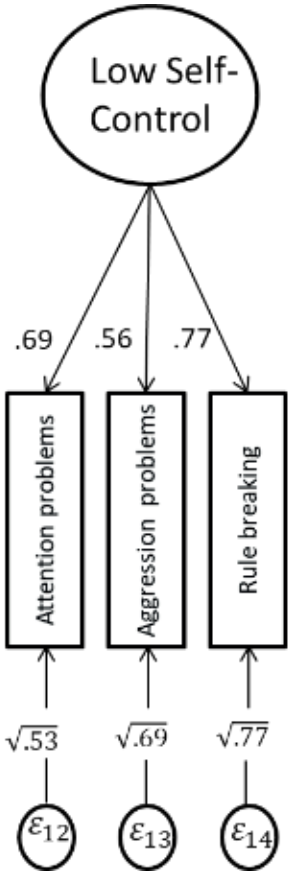
To conclude, most adolescents develop well and find their way into society without many problems, but not all adolescents do. Ill decisions and reckless behaviors due to low self-control in adolescence are concurrently and longitudinally costly (Caspi et al., 2016; Moffitt et al., 2011), and revealing possible factors contributing to individual differences in self-control is necessary. Applying a genetically sensitive design, this study points to the existence of a directional effect, in the presence of a genetic correlation, of family conflict on low self-control in adolescence. Future intervention and prevention practices should take this mechanism into account, when aiming to target adolescents at risk for developing low self-control.

SUPPLEMENTS

CHAPTER 6



Supplement 1. Dimensionality of Family Conflict



Supplement 2. Dimensionality of Self-Control

Supplement 3. Decomposition of Residual Errors, and Covariance Between Residuals of "Hitting" and "Swearing", Into Genetic and Environmental Variance (See Figure 3)

Trait	Measure	A	C	E
Family Conflict	Everyone involved	.28 (.14, .42)	.03 (-.07, .14)	.69 (.63, .74)
	Hitting	.47 (.43, .51)	.00 (-.01, .01)	.53 (.49, .57)
	Criticizing	.23 (.06, .39)	.05 (-.07, .16)	.73 (.66, .79)
	Often quarrel	.23 (.03, .44)	.03 (-.12, .19)	.74 (.66, .82)
	Swearing	.57 (.51, .63)	.00 (.00, .00)	.73 (.67, .80)
	Same person fights	.27 (.07, .48)	.01 (-.12, .14)	.72 (.63, .80)
	Visibly angry	.19 (.12, .26)	.00 (.00, .00)	.81 (.74, .88)
	Quarrel during bedtime	.20 (.05, .36)	.09 (-.03, .20)	.71 (.65, .77)
	Conflicts daily basis	.24 (.10, .39)	.05 (-.06, .15)	.71 (.66, .77)
	Throwing things household	.05 (-.14, .23)	.30 (.16, .45)	.65 (.58, .72)
	Always conflict during dinner	.08 (-.14, .29)	.29 (.12, .45)	.64 (.56, .72)
	Covariance "Hitting" and "Swearing"	.30 (.23, .38)	-	.70 (.65, .77)
Self-control	Attention problems	.25 (.19, .31)	-	.75 (.69, .81)
	Aggression problems	.23 (.17, .29)	-	.77 (.71, .83)
	Rule breaking	.19 (.14, .25)	-	.81 (.75, .86)

Note: For the final model we fitted an ACE model for family conflict and an AE model for self-control. As a result, we decomposed residual errors for family conflict into A, C, and E while for self-control we decomposed the residual errors in A and E.

Supplement 4. Model's syntax

see https://www.sciencedirect.com/science/article/abs/pii/S0890856719301868

Chapter 8

Stressful life events and self-control: Testing gene x environment interaction with a polygenic approach

ABSTRACT

Ill decisions and reckless behaviors due to low self-control are concurrently and longitudinally costly, and revealing possible factors contributing to individual differences in self-control is necessary. It is hypothesized that genetically sensitivity interacts with life stressors in the prediction of the development of low self-control (gene environment interaction), yet attempts to test this hypothesis mostly concern candidate gene studies yielding inconclusive results. The goal of this research was to bring findings from large scale gene identification studies into the developmental psychology framework, taking the polygenic nature of complex traits into account. Using data of a large population-based twin sample, we tested whether polygenic risk scores for self-control problems – based on the most recent ADHD GWAS – predict self-control problems in adults, and whether this polygenic risk scores interact with the presence of environmental stressors. While polygenic scores and life stressors significantly predicted low self-control, we did not find a significant interaction effect. Future recommendations for research on G x E in the etiology of self-control will be discussed.

Keywords: self-control, life stress, polygenic score, gene environment interaction, diathesis stress, GWAS

Based on: Willems, Y. E., Hottenga, J. J., Ligthart, L., Willemsen, G., Boomsma, D. I., Finkenauer, C., & Bartels, M.(submitted). Stressful life events and self-control: Testing gene x environment interaction with a polygenic approach.

Not being able to exert self-control – the inability to alter unwanted impulses and behavior, in order to bring them into agreement with goal-driven responses – places individuals at risk for myriad negative outcomes (de Ridder, Lensvelt-Mulders, Finkenauer, Stok, & Baumeister, 2012). For example, individuals with low self-control do worse at school and work, display unhealthier lifestyles (more substance use, higher sugar intake), and are more likely to experience mental health problems than individuals with high self-control (Moffitt et al., 2011; Vazsonyi, Mikuska, & Kelley, 2017). Because low self-control is a powerful predictor of psychological and physiological problems, it is important to identify how individual differences in self-control in the population arise.

In a recent meta-analysis, we report that differences in self-control are for about 60% accounted for by genetic differences and for 40% by environmental factors (Willems, Boesen, Li, Finkenauer, & Bartels, 2019). A number of environmental factors have been associated with lower levels of self-control in the population such as socioeconomic disadvantage, dangerous neighborhoods, delinquent peers, and family violence (Duckworth, Kim, & Tsukayama, 2013; Hostinar, Ross, Chen, & Miller, 2015; Turner, Piquero, & Pratt, 2005; Willems, Li, Hendriks, Bartels, & Finkenauer, 2018). Theoretical explanations for this include that stressful environments hamper self-control development (Davies & Cummings, 1994), stress depletes psychological resources necessary to exert self-control (Finkenauer et al., 2015) , and/or that it is not beneficial to show self-control in stressful contexts (Ellis, Bianchi, Griskevicius, & Frankenhuis, 2017).

While most studies thus far focus *either* on environmental *or* genetic factors, there is a growing number of studies highlighting the need to consider both environmental and genetic components and their interplay to understand individual differences in self-control (Cecil, Barker, Jaffee, & Viding, 2012; Willems et al., 2019b.). A frequently applied framework to understand this interplay is the diathesis-stress model, which proposes that stress may activate a vulnerability ("a diathesis") that transforms the potential of (genetic) vulnerability into the actuality of psychopathology (Monroe & Simons, 1991). This genetically driven sensitivity to environments proposed by the diathesis-stress model can also be operationalized as gene by environment interaction (G x E), where certain genotypes vary as a function of their sensitivity to the environment (Kendler & Eaves, 1986; Plomin, DeFries, & Loehlin, 1977).

Translating this theoretical framework to self-control, it is hypothesized that the genetic vulnerability for low self-control interacts with life stressors elevating the risk to actually develop low self-control (Belsky & Beaver, 2011; Kochanska, Philibert, & Barry, 2009). Attempts to test this hypothesis, however, mostly

concern candidate gene studies, yielding inconclusive results (Beaver, DeLisi, Vaughn, & Wright, 2010; Boisvert, Wells, Armstrong, & Lewis, 2018). Importantly, the reliability of candidate gene findings is widely debated as they are at odds with our knowledge that behavioral traits have a polygenic architecture being explained by effects of many variants of small magnitude across the genome rather than a handful of selected genes (Dick et al., 2015; Duncan & Keller, 2011). An approach that is in line with the polygenic structure of most human traits is a polygenic approach, which allows researchers to calculate a genetic score (a polygenic score, PS). PS represents a composite of additive effects of the multiple genetic variants associated with a trait (International Schizophrenia Consortium, 2009).

Recently, a number of studies use this PS approach to assess gene by environment interaction for mental health outcomes such as depression and schizophrenia (Colodro-Conde et al., 2018; Hatzimanolis et al., 2017; Peyrot et al., 2014). These studies are relatively novel, and the results have been mixed. For example, some G x E studies focusing on depression did not find a significant interaction between polygenic score for depression and personal life events (Peyrot et al., 2017), while others found that the interaction positively contributed to the risk of the actual development of depression (Colodro-Conde et al., 2018; Peyrot et al., 2014). This approach has not, however, been applied to the interaction between genetic vulnerability for low self-control and life stressors specifically. The goal of this study is test whether polygenic scores for self-control problems predicts low self-control, and whether polygenic scores interact with the presence of environmental stressors.

METHODS

Participants

Data for this study came from the Netherlands Twin Register (NTR), initiated in 1987 at the Vrije Universiteit (VU) Amsterdam. The NTR is a longitudinal, prospective study collecting data from twins, siblings and their parents, and sends out surveys every 2 – to 3 years on health, lifestyle, and personality (see Willemsen et al., 2013 for more details on data collection). We used an adult subsample of the NTR, with data available on self-control, experienced life stressors, and genotype (N=5,839, M_{age} = 41.14, SD_{age} = 15.45, all European Ancestry). All participants provided written informed consent, and the data collection was approved by the Central Ethics Committee on Research Involving Human Subjects of the VU University Medical Centre, Amsterdam.

Genotyping

Genotyping was performed at various points in time using multiple platforms following manufacturers protocols. After genotyping, similar quality control steps were performed within each array: individuals were excluded if (1) the genotyping call rate was below .90, (2) if there was a mismatch between the DNA - and expected sex, or (3) they had a Plink heterozygosity F statistic outside the -0.10 - 0.10 range. Additionally, we excluded single-nucleotide polymorphism (SNPs) if (1) the minor allele frequency (MAF) was less than .01, (2) the call rate was less than .95, (3) the number of Mendel errors exceeded 20, (4) they differed in allele frequency with the GONL reference set V4 with more than 0.10, if (5) they were palindromic with MAF > 0.40, or (6) the *p*-value of the Hardy-Weinberg equilibrium (HWE) test was less than $1x10^{-5}$ (Fedko et al., 2015; Manichaikul et al., 2010; Purcell et al., 2007).

Based on ~10.6k overlapping SNPs between platforms, IBD was calculated between all genotyped individuals and samples were removed if they did not fit the expected familial relations. For each array, the non-overlapping SNPs across all platforms were cross-imputed with the GoNL reference set V4 using the MACH-ADMIX software. Then SNP QC was redone, and SNPs were removed if they showed association with platform (P < 0.00001), if the imputation quality R2 <0.90, and if Mendel error rate < mean+3sd. The QCed data were then used for a second round of imputations to 1000 Genomes phase 3 version 5 on the Michigan imputation server (1000 Genomes Project Consortium, 2015; Deelen et al., 2014; Liu, Li, Wang, & Li, 2013). Meanwhile the cross-chip imputed data were also used to identify non-European ancestry with genetic Principal Components Analysis (PCA), projecting 10 principal components from the 1000 genomes reference set on the NTR data (Price et al., 2006). Afterwards we excluded individuals with principal component values other than Dutch and/or British populations, and recalculated 10 principal NL components to capture the variation within the Netherlands (Abdellaoui et al., 2013).

For this study the European samples, imputed to 1000G phase 3v5 were used for polygenic scoring. Extra SNP filters for scoring were applied these data, namely only ACGT SNPs on the autosomes, no SNPs with duplicate positions, no SNPs with 3 or more alleles, MAF > 0.01, HWE p > 10 -5 and genotype call rate > 0.99, leaving 7,411,699 SNPs.

Polygenic score

A polygenic score is based on SNPs that are related to a certain outcome. Genome Wide Association Studies (GWAS) use genetic data of large samples to find such relevant SNPs, and provide publicly accessible summary statistics for the effect size of specific SNPs on the trait of interest. Using such summary statistics allows to create an individual polygenic score for all genotyped participants, weighing the predictive value of each individual SNPs and generating an overall predictive 'genetic score' (GS, Dudbridge, 2013). As such, with a polygenic score, you (partly) capture someone's genetic propensity for a trait by summing all risk alleles the person has weighted by the effect sizes estimated in the GWAS.

There is no GWAS specifically for self-control, so we created a polygenic score based on a GWAS on a trait closely related to self-control, namely the most recent attention deficit hyperactivity disorder (ADHD) GWAS (Demontis et al., 2019). While self-control more broadly reflects the overall conscious capacity to regulate impulses, emotions, and behaviors and focus on goal-oriented behaviors, both ADHD and self-control tap into the difficulties to control your impulses (Nigg, 2017). As the genotyped participants of the NTR were part of the ADHD GWAS, we used summary statistics where the NTR participants were left out to avoid an overestimation of the effects.

LD-pred was used to adjust the summary statistics polygenic score weights, taking linkage disequilibrium (LD) among SNPs into account (Vilhjálmsson et al., 2015), and modelling the LD using the a 2nd degree unrelated set of 2500 1000G imputed NTR individuals. Scoring was done using Plink 1.9 for the NTR target sample of 5,839 individuals. In line with earlier work on cognitive traits and psychiatric disorders, we set the fraction of causal markers to be .30 (Abdellaoui et al., 2018; Hugh-Jones, Verweij, St. Pourcain, & Abdellaoui, 2016; Vilhjálmsson et al., 2015).

Self-control

We used the ASEBA Self-Control Scale (ASCS) to assess self-control. This questionnaire has been elaborately validated in children (age 7 – 12) and adolescent samples (age 14 - 16, see Willems et al., 2018a). The ASCS consists of 8 items assessing varying dimensions of self-control (e.g., "I fail to finish things that I start", "Sudden changes in moods or feelings") measured on a 3-point scale (1= *Not true*, 2= *Somewhat* or *Sometimes True*, 3= *Very True* or *Often True*). Higher scores reflect overall lower levels of self-control.

Extending this earlier work (Willems et al., 2018a), we examined the validity of the scale in adult samples in an independent NTR sample of participants without genetic data (*N*=7,523, so not included in the overall G x E analyses). The ASEBA, on which the ASCS is based, is particularly promising as its items are similar in content across informants and across age. As such, we selected the same 8 items as the ASCS in children and adolescents and examined whether the psychometric properties of the ASCS are satisfactory in adults. Results showed a similar factor structure of the ASCS in adulthood as in childhood and adolescence, with a decent Cronbach alpha coefficient (.70), and good test-retest reliability (the correlation between the ASCS at age 16 and the ASCS age 18-25 was .58, 95% CI [.53,.63], *N*= 584), indicating the scale is psychometrically sound in adult samples.

Stressful life events

We used the Dutch Life Event Scale ("Schokverwerkings Inventarisatie Lijst", Van der Velden et al., 1992) to assess the experience of stressful life events across the lifespan. In line with previous work (Middeldorp et al., 2010; Peyrot et al., 2013), we included the following experienced stressful life events: traffic accident, violent assault, sexual assault, robbery, serious illness or injury of self or a significant other (e.g. partner, child, parent), death of a significant other, dismissal from work, financial problems, and relationship problems with partner (Middeldorp et al., 2010). Response categories included 1= *Not experienced*, 2= *Experienced Less than a year ago*, 3= *Experienced 1-5 years ago*, 4= *Experienced longer than 5 years ago*. We created a sum score for the number of life events experienced in the previous year and a sum score for the number of life events across the lifespan, respectively. Important to note is that this scale only assesses negative life events, and not positive life events which can be stressful as well (e.g. marriage, birth of child, etc.).

Statistical analyses

Regression analyses were carried out with self-control as the dependent variable, using generalized estimation equations (GEE) in SPSS (version 25.0, IBM Corp, 2017). We standardized all the variables and analyzed main effects (stressful life events and polygenic score, respectively) and the interaction effect (stressful life events x polygenic score) in one model. We did these analyses separately for life events in the previous year and life events over lifetime (resulting in 2 tests). Applying Bonferroni-type adjustment, we divided p <0.05 by the number of tests on the dependent variable (2 tests in

total), taking p <0.025 as level of significance. To correct for the dependency between participants (i.e., family members), we allowed for correlated residuals between members of the same family, and tests were based on applied robust sandwich-corrected standard errors (Abdellaoui et al., 2018; Rebollo, de Moor, Dolan, & Boomsma, 2012). To avoid the findings being driven by confounders rather than by the genetic or environmental variables of interest, we added age, age^2, sex, 10 principal components, and array as covariates. Doing so does not control for the possible effect these confounders have on the G x E interaction, possibly resulting in attenuation of the G x E estimates. Therefore, we also included interaction-terms of covariates x genes (e.g., age x polygenic score) and covariates x environment (e.g., age x stressful life event) to eliminate effects of interactions between covariates and the variables of interest (Keller, 2014).

RESULTS

The prevalence of experienced life events in *the last year* ranged between 0 and 5, and the prevalence of experienced life events over *the lifetime* ranged between 0 and 13 (see Table 1). In Table 2, we report the standardized betas, standard errors, and p-values of the main and interaction effects. There was a significant main, albeit small, effect of the polygenic score on low self-control, and a significant main effect of life events experienced in the last year. The interaction between polygenic score and negative life events in the last year, however, did not significantly predict low self-control. We report similar test statistics for life events experienced over the lifetime. While there was a significant main effect of polygenic score and experienced life events experienced over the lifetime, respectively, the interaction between polygenic score and negative life events experienced in the life time did not significantly predict low self-control.

Perhaps, it is gene-environment correlation that is applicable rather than gene-environment interaction that plays a role and attenuating our findings. As an exploratory analyses, we correlated our polygenic score (for ADHD) and the environment (negative life events past year, negative life events across the life time). Doing so resulted in we find significant albeit small correlations (NLE past year r=.022, p=.016, NLE life time r=.027, p=.004).

Table 1. Percentage of participants (absolute numbers in brackets) who experienced negative life events last year and over the lifetime

Number of negative life events (NLE)	% (N) NLE experienced last year	% (N) NLE experienced over the lifetime
0	68,8 (4017)	12,2 (712)
1	23,6 (1378)	24,0 (1401)
2	5,7 (333)	23,1 (1349)
3	1,4 (82)	16,1 (940)
4	0,4 (23)	10,7 (625)
> 5	0,1 (6)	13,9 (812)

Table 2. Coefficients of main and interaction effects of negative life events (NLE) and polygenic score (PS), predicting self-control, with for self-control higher scores mean lower self-control.

	β	Std. Error	95% CI	*p*-value
Main effects				
PS ADHD	.033	.014	[.008, .059]	.011
NLE previous year	.128	.015	[.098, .158]	<.001
Interaction effects				
PS * NLE *previous year*	.012	.014	[-.015, .040]	.378
Main effects				
PS ADHD	.030	.013	[.005, .056]	.021
NLE over the lifetime	.134	.014	[.107, .161]	<.001
Interaction effects				
PS * NLE over the lifetime	-.010	.014	[-.034, .019]	.589

DISCUSSION

In line with the diathesis-stress or gene-environment (G x E) interaction theories, we hypothesized that genetic risk interact with experienced life stressors thereby propelling individuals into the development of lowered self-control. In this research, we aimed to test this hypothesis. We used data of large set of genotyped adults of the Netherlands Twin Register (*N*=5,839), including polygenic scores based on an ADHD GWAS (Demontis et al., 2019), self-reports on self-control, and self-reported experienced life stressors in the past year and over the lifespan. While we found small main effects of life

stressors and polygenic scores on low self-control, respectively, we did not find evidence for an interaction effect between genetic risk (polygenic score) and life stressors (in the last year nor in the lifespan) in the prediction of low self-control.

The findings are interesting in a number of ways. First, we found main effects of both genetic and environmental effects on self-control. Specifically, the latter is in line with the increasing line of research postulating that contextual stressors explain individual differences in self-control (Willems, 2019b). Second, the hypothesis that genetic risk interacts with stressful environments is theoretically appealing as it provides a clear framework how "nature" and "nurture" collaboratively influence individual differences within the population. However, our results, and those of others (Assary, Vincent, Keers, & Pluess, 2018; Keller, 2014; Peyrot et al., 2018), highlight that finding empirical evidence for this hypothesis is difficult, and we need to critically think how we can methodologically capture this complexity. Second, a non-significant finding does not necessarily equal evidence for the non-existence of an effect, and replication of our study is recommended (Shrout & Rodgers, 2018). A great lesson we have learned from the candidate gene studies is that replication is key, and that publishing significant and non-significant results are necessary to paint a full picture the way in which G x E explain individual differences in the population (Yong, 2019).

Third, an alternative hypothesis is that we should focus on gene-environment correlation rather than gene-environment interaction, this as life events can be explained by familial factors (Middeldorp, Cath, Vink, & Boomsma, 2005). Gene-environment correlation reflects the correlation between a person's genotype and the environment he/she grows up in (passive rGE, or a correlation between a person's genotype and the environment he/she selects or elicits (evocative or active rGE, Kendler & Eaves, 1986; Plomin et al., 1977). For future research it would be promising to use twin- and adoption designs to investigate the way in which gene-environment correlations explain self-control differences in the population (Distel et al., 2011; Middeldorp, Cath, Beem, Willemsen, & Boomsma, 2008). Importantly, In case we did find a significant gene x environment interaction, we should have interpreted the results with caution due to the possible confounding effects of gene environment correlation (Kendler & Eaves, 1986).

The results of the current study should be interpreted with some limitations in mind. As there is no GWAS on self-control, we used the GWAS of ADHD (Demontis et al., 2019) thereby not fully capturing the broader dimensions of

the self-controlling capacities as revealed in the small effect size of the main effect. Self-control is a heterogeneous construct, both theoretically (Nigg, 2017) and empirically (Duckworth & Kern, 2011), and in the future we need to think how to best genetically capture self-control. A promising development in the field is Genomic Structural Equation Modeling (Grotzinger et al., 2019), using publicly available GWAS data to model multivariate genetic associations among phenotypes. One way forward would be to test whether this method can reliably create polygenic scores self-control, using correlations between different related traits (e.g. ADHD, conscientiousness, executive functioning) to generate a more accurate polygenic score. Additionally, life events only included negative life events (e.g., theft, illness, financial strain etc.) and not positive life events (e.g., marriage, birth of a child) which can also impact people's self-control levels (Bleidorn, 2015; Pronk et al., 2019). Future research taking more fine-grained life events into account could be promising. Moreover, while we included adults in this study, it would be interesting to apply similar analyses in childhood to generate insights in the ways in which gene-environment interplay play are comparable in adults and children.

In sum, we tested the hypothesis that genetic risk interacts with life stressors in explaining individual differences in low self-control in the population. We did not find evidence to support this hypothesis. We hope this article will stimulate future research on this important topic integrating influences of both "nature" and "nurture", an area we expect to further develop in the years to come.

Chapter 9

Summary and General Discussion

Health researchers benefit from a better understanding of what propels some people into positive- and others into negative health trajectories. People's self-control has been coined as a promising study target as ill decisions due to low self-control are at the cost of physical and psychosocial functioning on the short - and the long run (Caspi et al., 2016; de Ridder, Lensvelt-Mulders, Finkenauer, Stok, & Baumeister, 2012; Moffitt et al., 2011). The risks of low self-control in a variety of life domains raises the question: **What factors explain differences in self-control in the population?** Finding answers to this question was the aim of this dissertation. First, we meta-analyzed the literature to summarize environmental and genetic influences on self-control. Second, using data of the Netherlands Twin Register (NTR), we explored causes of self-control differences while taking the interplay between genetic and environmental factors into account. In this chapter, we summarize our results, discuss the implications of our findings, and highlight research opportunities for the future.

Meta-Analyzing the Literature

Intuitively, we think of the family context when pinpointing environmental factors related to self-control. Not surprisingly, much research focused on the link between parenting and self-control, especially during early and middle childhood. However, there is surprisingly little consensus regarding the presence of this link during adolescence, a phase when contexts outside the household become increasingly important. In *Chapter 2* we therefore conducted a large-scale **meta-analysis quantifying the association between parenting and self-control across adolescence**. We synthesized the results of 191 studies and observed a small to moderate association between parenting dimensions (positive parenting, negative parenting, parent-child relationship) and self-control. The results suggest that more positive parenting and better parent-child relationship coincide with higher self-control, while negative parenting coincides with lower self-control in adolescents. The associations were stable across countries, age of adolescents, and adolescent gender. A few methodological factors moderated the relationship, such as type of informant and whether parenting and self-control were assessed by the same person. Unique about this meta-analysis was our focus on both parent- *and* child-driven effects. Interestingly, the overall effect size from adolescent self-control to parenting (child-driven effects) was not significantly different from the overall effect size from parenting to adolescent self-control (parent-driven effects).

Because the literature posed family violence as a particular risk factor for self-control development (Finkenauer et al., 2015), we conducted a meta-analysis to quantify their association in *Chapter 3*. We synthesized the results of 27 published studies, including 143 effect sizes. Overall, we found a small to moderate negative association between family violence and self-control. This association decreased with age and was smaller in longitudinal studies as compared to cross-sectional studies. The association was stable across gender, country, and informants. This implies that family violence and low self-control co-occur, especially in early adolescence.

Together the findings of *Chapter 2* and *Chapter 3* imply that, while adolescents spend less time in the household with family members and hang out more with peers, generally **parenting continues to be associated with the self-control** of adolescents. Importantly, children and adolescents are not passive recipients to their environment, and their self-control influences the parenting style of their parents, in turn. There are transactional processes taking place where parents influence adolescent self-control and, vice versa, adolescent self-control results in certain parenting practices. Together, this highlights that the general assumed direction of effects from parents to children is too simple.

To paint a more complete picture of factors shaping self-control, we aimed to extend our work on contextual factors (parenting in *Chapter 2* and family violence in *Chapter 3*) by quantifying the overall heritability of self-control. In *Chapter 4*, we therefore synthesized 31 twin studies, and meta-analyzed monozygotic and dizygotic twin correlations to calculate the heritability of self-control. We found an **overall heritability estimate of 60%,** with the remaining 40% of the variance explained by the unique environment and measurement error. This heritability estimate was the same for boys and girls and across age but was higher for parent reported self-control than self-reported self-control.

This implies that individual differences between individuals in their self-control capacities are for 60% explained by genetic differences between these individuals. It is important to bear in mind that a heritability estimate is probabilistic not deterministic (Johnson, Turkheimer, Gottesman, & Bouchard, 2009). It suggests that it is likely that for some individuals it is easier to exert self-control than for others, even when exposed to the same intervention or environment, and this is **partly explained by their genetic make-up** (Harold, Leve, & Sellers, 2017). Overall, when aiming to understand the origins of self-control differences in the population, we should not only take the context (e.g., parenting) but also genetic differences into account.

Interplay between Genetic and Environmental Factors

Individual differences in the population can be explained by a multitude of factors, ranging from differences in environmental exposures [*Chapter 2, Chapter 3*], to differences in genetic variations [*Chapter 4*]. Historically, the debate pitted a socialization perspective, which considers individual differences as rooted in environmental exposure, against a biological perspective, which considers individual differences as originating from genetic influences (Tucker-Drob & Bates, 2016). By now, we know that genetic and environmental influences are not mutually exclusive or additive per se, and part of the variation in the population is the result of the *interplay* between the two. Especially when aiming to distill directional effects between the family context and a child's behavior, it is important to take the **gene-environment interplay** into account (D'Onofrio, Lahey, Turkheimer, & Lichtenstein, 2013; Johnson et al., 2009). Namely, the association between the family context and child outcomes can be the result of a true directional effect or, alternatively, be caused by common genetic factors simultaneously influencing both the family context and child outcomes (genetic pleiotropy or genetic confounding, Pingault et al., 2018). Not taking this alternative pathway into account potentially confounds research findings, hindering an attempt to reveal causal mechanisms explaining the outcome. As such, it is important to further test the associations we found between the family context and self-control *[Chapter 2, Chapter 3]*, while taking environmental *and* genetic factors into account *[Chapter 4].*

The wealth of data from the NTR provided us with the unique opportunity to create and validate a self-control scale. In *Chapter 5*, we showed the potential of the Achenbach System of Empirically Based Assessment (ASEBA) to assess self-control. We selected 8 items, similar in content across age and informant, and tested the validity of the **ASEBA self-control scale (ASCS)** across childhood and adolescence. We found good internal consistency and moderate to strong correlations (1) between the ASCS and outcomes theoretically related to self-control (e.g., educational performance, wellbeing, substance use), (2) across different informants (e.g., mother-, father-, teacher-, self-report), and (3) across time points (e.g., from age 7 to age 16). Additionally, we found heritability estimates corresponding to earlier studies (around 60%, see *Chapter 4*). In *Chapter 9*, we demonstrated that this scale is also psychometrically sound in adults.

The validity of the **ASCS across the lifespan provides a wide array of opportunities** for researchers to further investigate the origins of individual differences in self-control. First, the scale was validated for parent-, teacher-

and self-reports, allowing researchers to assess self-control across multiple contexts and informants. Second, because low self-control in childhood is a predictor for long term self-control problems and related adverse life outcomes (Caspi et al., 2016; Duckworth, Tsukayama, & Kirby, 2013; Moffitt et al., 2011; Tsukayama, Toomey, Faith, & Duckworth, 2010), using the scale early in life could potentially aid in the detection of children at risk. Third, the scale provides opportunities for secondary data-analyses. The ASEBA is an internationally widely applied scale; multiple large longitudinal family-studies have ASEBA data readily available (e.g., EGDS, TCHAD, TRAILS, Leve et al., 2013; Lichtenstein, Tuvblad, Larsson, & Carlström, 2007; Ormel et al., 2012). The ASCS allows those research groups to assess self-control in existing data and may also facilitate new international collaborative efforts investigating the causes and consequences of self-control across the lifespan. For example, aggregating such data would allow for cross-cultural assessments (e.g., comparing self-control predictors between countries), generation comparisons (e.g., comparing levels of self-control in youth from the 1980's and youth from the 2000's), fast replication (e.g., validating the findings in multiple datasets), and development of statistical methods (e.g., making use of the large sample sizes).

The validation of the ASCS allowed for further investigation of the link between family factors and self-control as shown in *Chapter 2* and *Chapter 3* in the large and genetically informative NTR family data. In *Chapter 6,* we sought to investigate whether **family connectedness and self-control** are causally related taking both genetic and environmental influences into account. We found a significant, but small, phenotypic association, suggesting that adolescents who experience more family connectedness report higher levels of self-control across adolescence. The nature of this association was mainly explained by common genetic factors as in monozygotic twin pairs (who share 100% of their genetics and family environment), the twin who experienced more family connectedness did not show higher self-control and, vice versa, the twin showing higher self-control did not experience more family connectedness. This implies that when interpreting results of correlations between these two traits in analyses not taking family relatedness into account, we should be cautious in the interpretation of these associations as causal.

In *Chapter 7,* similar to *Chapter 6*, we investigated the link between **family conflict and self-control** during adolescence while taking both genetic and environmental factors into account. Here we tested whether the association between family conflict and self-control is explained by: 1) common genetic factors, 2) a bi-directional influence between family conflict and self-control,

3) a unidirectional association with family conflict predicting low self-control, or 4) a unidirectional association with low self-control explaining family conflict. Applying the 'Direction of Causation' twin model, we demonstrated a directional effect of family violence on lowered self-control over and above mutual genetic influences. So, while the link between family connectedness and self-control was likely non-causal in nature, the link between family conflict and self-control is more likely to reflect a directional effect. Implications of this result include that researchers and practitioners can **expect low self-control in the wake of family violence** and should therefore not treat them as separate problems. Targeting family violence could potentially break the vicious circle of maladaptive self-control development.

With twin studies, we discuss to what extent differences in the population are explained by environmental or genetic variance. For example, twin models allow us to investigate the overall heritability of a trait (see *Chapter 3, 4*), or to investigate the role of the environment on an outcome while controlling for genetic confounding (see *Chapter 6*, *7*). These models do not, however, use information on specific genetic variants that are involved in a trait or the co-occurrence between traits. In the field of molecular genetics, there is a focus on genetic variants, especially single nucleotide polymorphisms (SNPs), that are associated with behaviors, applying analyses such as genome wide association studies (GWAS). The aim of *Chapter 8* was to move beyond heritability and use molecular genetic information to further investigate gene-environment interplay. In line with the diathesis-stress theory, it is hypothesized that someone's genetic risk interacts with environmental stressors as a shaping factor for the development of self-control problems (Monroe & Simons, 1991). This is also referred to as **gene-environment interaction** (G x E), that is, the magnitude of the genetic influence varies as a function of an environmental exposure (Kendler & Eaves, 1986; Plomin, DeFries, & Loehlin, 1977). Thus far, however, this hypothesis has mostly been tested using candidate gene studies, and different approaches are necessary to take the polygenic nature of complex traits such as self-control into account (Dick et al., 2015; Duncan & Keller, 2011). Accordingly, in *Chapter 8,* we examined whether individuals with an increased genetic liability to develop ADHD, based on polygenic scores, who also experienced more life stressors showed more self-control problems (i.e., an interaction effect) than individuals who only have either a high polygenic score or experienced more life stressors. While we found small main effects for polygenic risk scores and life stressors on low self-control, we did not find a significant interaction effect on self-control.

The diathesis-stress or gene-environment interaction hypothesis is theoretically appealing as it provides a vivid framework how "nature" and "nurture" collaboratively explain the origins of self-control differences in the population. Namely, it could explain why, even when experiencing the same environmental exposure, some individuals will develop health problems while others do not. Empirically, however, **finding statistical evidence for this hypothesis remains a challenge**, and more research is needed to investigate how to better detect G x E. Still it remains important to critically think whether more efforts are necessary to methodologically capture this complexity or, alternatively, whether we have to revisit our theories considering the applicability of G x E to self-control. Perhaps we should focus on gene-environment correlation (the correlation between the genotype you inherit, and the environment you experience) rather than gene-environment interaction. A person's level of self-control (which is partly genetic) influences the way in which they perceive their environment, or seek out certain environments, explaining how both their genetic propensity and life stressors are correlated and result in certain life outcomes.

Future Directions

While this dissertation provides some answers, it also raises new questions concerning the way in which self-control differences in the population arise. Here we will highlight some **questions for future research** and propose methods that can be used to answer them.

How do we go beyond correlations in meta-analyses? Meta-analyses are a useful and popular tool to systematically review the scientific literature and to quantify an overall association between trait X and Y. They have the capacity to compare and contrast results from different individual studies, revealing patterns that only come to light with increasing power when pooling multiple studies. However, it is important to note that the results of our meta-analyses in *Chapter 2,3* & *4* reflect correlations, not necessarily causation. To better understand what mechanisms underlie human behavior, it is important to capture *change*: we do not only want to know whether X and Y are associated but also if X truly explains the difference in Y over time (and vice versa, Heise, 1970). To assess change, we need to take autoregressive effects into account (i.e. stability of traits over time) and apply more fine-grained analyses to investigate longitudinal associations. One way forward would be to meta-analyze not only cross-sectional and longitudinal effect sizes, but also autoregressive effects, modelling associations *and* change over time. New

methods are underway to do so, promising future studies to conduct cross-lagged panel models while pooling data of multiple individual studies (Cheung, 2015b).

How do we gain insights in the molecular genetic basis of self-control? While in *Chapter 4* we highlight the overall heritability of self-control, we do not know which specific set of genetic variants are related to self-control. Heritability estimates and the use of twin models are particularly promising to understand the underlying mechanisms explaining the overlap between two traits (e.g., family conflict and self-control, *Chapter 7*). However, taking it a step further by identifying the genetic variants is important, as improved molecular information potentially improves our ability to predict who is at an increased risk to develop self-control problems. Additionally, it allows us to advance our investigation of gene-environment interaction, something that was limited in our study by the lack of proper molecular genetic instruments that are specific to self-control (i.e.. in *Chapter 8* we used the polygenic score for ADHD as a proxy, which showed low predictive value). A natural extension of this dissertation would therefore be to investigate the molecular genetic etiology of self-control. There are a number of ways to do so. First, an international consortium on self-control should be initiated, stimulating the collaboration between groups that have both genotype data and measures of self-control (e.g., see Boomsma et al., 2015). Considering the wide use of the ASCS *[Chapter 4]* in large family-based research cohorts, initiating a self-control consortium could be promising approach for the future. Second, the newly developed multivariate genome-wide-association meta-analysis (GWAMA) could be applied (Baselmans et al., 2019). This allows scientists to analyze a multitude of self-control related traits, increasing statistical power to detect genetic variants that are associated with self-control, while capturing a broad spectrum of traits tapping into self-control capacities (a GWAS of 'the self-control spectrum'). Third, applying Genomic SEM could be promising as it allows to infer genetic information of an unmeasured or heterogeneous trait (i.e. self-control) using genetic information of measured traits that are related to self-control (e.g. educational attainment, conscientiousness, risk taking, executive functioning, Grotzinger et al., 2019). However, we should keep in mind that it remains a challenge to determine how to best theoretically conceptualize or empirically measure self-control (see "Conceptualizing Self-control: It's Complicated", *Chapter 1*). So while these three methods seem promising to gain insights into the molecular etiology of self-control in the future, they can only come to fruition when going hand-in-hand with improving

the integration of self-control related concepts among investigators (Nigg, 2017).

How do we make our samples more diverse? In all our chapters, we included samples mainly based on participants from the U.S.A. and the Netherlands. Replicating our findings across different populations in the future is key. Especially for molecular genetic research, it is important to consider more diverse samples (e.g., conduct GWAS studies in various populations, Gross, 2018). While more diverse molecular genetic samples are underway (Hyman, 2018), there needs to be an increasing effort to avoid genomic opportunities being a benefit only for certain populations. This is not only a challenge and an important topic for molecular geneticists, but also for researchers in the field of parenting. For example, when taking a closer look at our parenting meta-analyses, we see that most of the studies were conducted in the U.S.A.. Providing open access to our data and scripts, we hope to stimulate future research to update our work with more diverse samples and with studies not published in English. Similarly, in our twin analyses, we included data of a population-based family cohort, in which we know that high risk families less frequently participate and/or drop out earlier (Wolke et al., 2009). Heritability estimates, however, depend on the population included. For example, in societies with large disparities in the access to high-quality education, the heritability for cognitive abilities is larger in higher socioeconomic contexts as compared to lower socioeconomic contexts (Harden, Turkheimer, & Loehlin, 2007; Tucker-Drob & Bates, 2016). Consequently, efforts to think about how to include those at-risk families in our research is important in the future, if we want to gain better insights into mechanisms propelling families into negative socio-emotional and health trajectories.

How do we improve our understanding of the causes of self-control? Understanding causality is another key to future research, because only when revealing causal factors we know what mechanisms could be targeted in order to realize change. Doing so, however, is a complex endeavor. In this dissertation, we applied multiple models to investigate causality (e.g., twin difference models, direction of causality model), yet considerably more work needs to be done to further map causes of lowered self-control. The methodological toolbox investigating causes of individual differences is rapidly increasing, providing promising avenues for future research (Pingault et al., 2018). For example, with the drop in genotyping costs and the methodological advancements in molecular genetics, Mendelian Randomization (MR) is becoming increasingly popular (Smith & Ebrahim, 2003). However, one key assumption of MR is

that there is no pleiotropy (i.e., no correlation between the genotype and the outcome, only a correlation between the genotype and the exposure), something that is difficult to ascertain. Of particular promise is therefore the recently developed MRDoC model, integrating Mendelian Randomization (MR) and Direction of Causation twin model (DoC, Minică, Dolan, Boomsma, de Geus, & Neale, 2018). In comparison to more traditional MR approaches, this method allows researchers to better incorporate genetic effects (by including polygenic scores), while more accurately testing for pleiotropy (using twin designs). Another interesting, recently proposed approach is the integration of polygenic scores in network modeling (Isvoranu et al., 2019). According to the network approach, the co-occurrence between two traits (e.g., self-control and health outcomes) is the result of a network of symptoms that directly influence one-another rather than the result of a latent variable causing the constellations of symptoms (Cramer, Waldorp, van der Maas, & Borsboom, 2010). Integrating these two methods (polygenic scores and symptom networks) allows to better identify pathways through which the combination of genetic risk factors increases (or decreases) the liability to develop a certain outcome. However, a key condition for these methods is having improved molecular insights into self-control. While the field of molecular genetics is developing at a high pace (Visscher, 2017), for most traits there still is a gap between the heritability of traits as postulated by twin studies and genetic prediction based on GWAS studies ("the Missing Heritability", Manolio et al., 2009). So, while both Mendelian Randomization and the network approach could be exciting opportunities in the future, it remains a challenge how to make best use of them in a time when we are still sorting out how to best molecularly capture complex traits.

How do we move from population estimates to individual based prediction? The results presented throughout this dissertation pertain to the population, not to the individual per se. However, we are entering a very exciting era where the increase in technological advancements allows us to move beyond population estimates and investigate processes at an individual level. The rapid progresses in molecular technologies (e.g., affordability genotyping), increasing use of real time measures of the environment (e.g., digital phenotyping), and advancements in computational capacity and algorithm development (e.g., machine learning), confer unprecedented power to understanding human behavior on the individual level (Darcy, Louie, & Roberts, 2016; Iniesta, Stahl, & McGuffin, 2016; Li, Li, Zhang, & Snyder, 2019). Ideally, in the future we can predict not only differences in self-control

on a population level, but also predict fluctuations in a person's self-control on a day-to-day level.

Conclusion

The title of this dissertation consists of two components. **"Out of Control"** refers to the individual differences in self-control we see in the population, with some people having more problems than others to stay in control. **"Causes of Individual Differences in Self-Control"** refers to our aim to understand which factors give rise to these individual differences. Using meta-analyses and twin designs, we showed that both environmental (parenting) and genetic factors (heritability estimate of 60%) play a significant role in explaining individual differences in self-control. Particularly, we see impairments of self-control in the wake of family violence. Practitioners and professionals should be aware that low self-control may result from the violence experienced at home and from the genetic transmission from parents to their children. In this dissertation, we highlight that investigating gene-environment interplay is highly complex, but necessary to understand causes of differences in self-control capacities. Examining the causes of self-control differences while taking gene-environment interplay into account remains an intriguing yet challenging area of research, which we expect to blossom in the years to come.

NEDERLANDSE SAMENVATTING

Zelfcontrole speelt een grote rol in ons dagelijks leven. We moeten ons concentreren op ons werk terwijl we worden afgeleid door sociale media, emoties reguleren bij het aangaan van nieuwe relaties en ongezonde snacks laten staan wanneer we op een dieet zijn. Zelfcontrole helpt ons met dit soort dagelijkse uitdagingen: het is de kracht om ons doen en laten onder controle te houden als er verleidingen op de loer liggen.

Niet iedereen heeft dezelfde hoeveelheid zelfcontrole. Sommige mensen vinden het erg lastig om zelfcontrole uit te oefenen, waardoor ze een groter risico hebben op talloze psychologische en fysiologische problemen. Mensen met weinig zelfcontrole lopen een groter risico op het verliezen van hun baan, vertonen vaker een ongezonde levensstijl (minder sport, meer obesitas, meer alcoholgebruik) en hebben meer kans op psychische problemen dan mensen met veel zelfcontrole (Caspi et al., 2016; de Ridder, Lensvelt-Mulders, Finkenauer, Stok, & Baumeister, 2012; Moffitt et al., 2011). Het is daarom belangrijk om factoren te identificeren die individuele verschillen in de mate van zelfcontrole verklaren.

Een veelvoud van factoren kan onze manier van doen bepalen, variërend van factoren op microniveau (bijvoorbeeld genen) tot factoren op macroniveau (bijvoorbeeld cultuur). Onderzoek doen naar deze factoren is complex, omdat ze zijn ingebed in individuen en contexten die elkaar wederzijds beïnvloeden (Bronfenbrenner, 1979). Lange tijd is er onderzoek gedaan naar contextuele invloeden, zoals de manier waarop familiefactoren individuele verschillen tussen mensen verklaren. In de laatste decennia, met de accumulatie van tweelingdata en de verlaagde kosten van DNA-testen, groeit de interesse in de rol van genetische invloeden op individuele verschillen. Terwijl in het verleden *nature* versus *nurture* het debat domineerde, is er tegenwoordig een toenemend besef dat *nature* en *nurture* gezamenlijk verklaren hoe individuele verschillen in de populatie ontstaan (Harold, Leve, & Sellers, 2017; Plomin, DeFries, Knopik & Neiderhiser, 2016).

Handige concepten om de wisselwerking tussen omgevings- en genetische factoren te begrijpen zijn gen-omgevingscorrelatie (rGE) en gen-omgevingsinteractie (G x E). Gen-omgevingscorrelatie beschrijft het proces waarbij iemands genotype samenhangt met de omgeving waarin hij/zij zich bevindt (Kendler & Eaves, 1986; Plomin, DeFries, & Loehlin, 1977). De associatie tussen opgroeien in een huishouden vol boeken en goed kunnen lezen is bijvoorbeeld niet noodzakelijk causaal. De associatie kan (gedeeltelijk)

verklaard worden door de samenhang tussen de genen die ouders doorgeven (bijvoorbeeld genen om goed te kunnen lezen) en de omgeving die ze creëren (bijvoorbeeld veel boeken in huis). Bij het onderzoeken van causaliteit is het daarom belangrijk om rekening te houden met gen-omgevingscorrelatie omdat dit mogelijk de relatie tussen blootstelling (het aantal boeken) en de uitkomst (goed kunnen lezen) beïnvloedt (D'Onofrio, Lahey, Turkheimer & Lichtenstein, 2013; Pingault et al., 2018).

Gen-omgevingsinteractie beschrijft het proces waarbij bepaalde genotypen variëren in hun gevoeligheid voor bepaalde omgevingen. Er wordt bijvoorbeeld verondersteld dat mensen een vergelijkbare stressvolle gebeurtenis kunnen ervaren, maar dat mensen met een genetische kwetsbaarheid meer kans hebben om psychische problemen te ontwikkelen als gevolg van deze blootstelling dan mensen met minder genetische kwetsbaarheid (Monroe & Simons, 1991).

Het is echter moeilijk om deze concepten empirisch te testen. Daarvoor zijn data nodig die zowel omgevings- als genetische informatie meet en statistische modellen moeten worden toegepast die met beide soorten informatie rekening kunnen houden (Jaffee, 2016). Hoewel er stappen in de juiste richting worden gezet (zoals het verzamelen van genetische data, de samenwerking tussen multidisciplinaire onderzoeksgroepen en de ontwikkeling van geavanceerde statistische modellen, Boomsma, Busjahn & Peltonen, 2002), zijn er tot dusver nog maar weinig studies die specifiek naar het dynamische samenspel tussen genen en de omgeving bij zelfcontrole hebben gekeken. Dit samenspel is echter belangrijk als we willen begrijpen hoe individuele verschillen in zelfcontrole in de populatie ontstaan.

Het doel van dit proefschrift is om factoren te identificeren die individuele verschillen in zelfcontrole verklaren. Het proefschrift bestaat uit twee delen. Het eerste deel onderzoekt de literatuur door systematisch na te gaan in welke mate omgevings- en genetische invloeden individuele verschillen in de mate van zelfcontrole verklaren. Het tweede deel richt zich specifiek op het *samenspel* tussen omgevings- en genetische factoren, en op de vraag of de causale factoren geïdentificeerd kunnen worden die verklaren waarom sommige mensen minder zelfcontrole hebben dan anderen.

Samenvatting van de literatuur

Veel onderzoekers richten hun onderzoek op de associatie tussen opvoeding in de vroege kinderjaren en zelfcontrole. Er is echter verrassend weinig consensus over de associatie tussen opvoeding en zelfcontrole bij adolescenten voor wie contexten buiten het gezin belangrijker worden (bijvoorbeeld school, vrienden,

eerste romantische relaties). Sommige onderzoekers stellen dat ouders alleen van belang zijn voor het ontwikkelen van zelfcontrole van hun kinderen wanneer ze jong zijn, terwijl andere onderzoekers stellen dat ouders een belangrijke rol blijven spelen tot laat in de adolescentie.

In Hoofdstuk 2 gebruiken we een grootschalige meta-analyse om de associatie tussen opvoeding en zelfcontrole tijdens de adolescentie te analyseren. In een meta-analyse wordt op basis van resultaten uit verschillende onderzoeken een robuustere uitspraak gedaan over een effect. Het voordeel van een meta-analyse is dat verschillende gemengde resultaten geanalyseerd kunnen worden. Daarmee kunnen hypotheses worden getoetst die niet te bevestigen zijn in een enkele studie. In Hoofdstuk 2 gebruiken we de resultaten van 191 studies. We vinden een significante associatie tussen opvoeding (positief oudergedrag, negatief oudergedrag, ouder-kindrelatie) en zelfcontrole. Deze associatie is constant tussen nationaliteiten, de leeftijd van adolescenten en het geslacht van adolescenten. Enkele methodologische factoren hebben een invloed op deze relatie: de associatie is sterker wanneer oudergedrag en zelfcontrole door de zelfde informant worden beoordeeld (bijvoorbeeld beide door de ouders of beide door het kind). Uniek aan deze meta-analyse is onze focus op mogelijke richtingen van het effect. Zo zien we dat oudergedrag een invloed heeft op de zelfcontrole van het kind, maar dat de zelfcontrole van het kind ook bepaald oudergedrag uitlokt.

In de literatuur wordt gesuggereerd dat familiegeweld een specifieke risicofactor is voor verminderde zelfcontrole (Finkenauer et al., 2015). Wij passen daarom in Hoofdstuk 3 een meta-analyse toe om de relatie tussen familiegeweld en zelfcontrole beter te kwantificeren. We includeren de resultaten van 27 gepubliceerde studies, en vinden een significante associatie. Deze associatie neemt af met leeftijd en is kleiner in longitudinale studies in vergelijking met cross-sectionele studies. De associatie is constant tussen nationaliteiten, het geslacht van adolescenten en informanten. Dit betekent dat gezinsgeweld en verminderde zelfcontrole samen voorkomen, vooral in de vroege adolescentie.

Samen impliceren de bevindingen van Hoofdstuk 2 en Hoofdstuk 3 dat oudergedrag significant geassocieerd blijft met de zelfcontrole van hun adolescenten, ook al brengen de adolescenten minder tijd door thuis met hun ouders en meer met vrienden. Belangrijk is dat kinderen en adolescenten geen passieve ontvangers zijn van hun omgeving. Ouders en kinderen beïnvloeden elkaar. Als ouder is het bijvoorbeeld veel makkelijker om liefdevol te reageren en constructieve feedback te geven aan een tiener die het goed doet op school

en op tijd thuis is na een nacht uitgaan, dan aan een tiener die veel spijbelt en stiekem alcohol drinkt. Dit benadrukt dat het algemeen veronderstelde idee dat ouders het gedrag van hun kind 'veroorzaken' te eenvoudig gesteld is, want kinderen 'veroorzaken' ook het gedrag van hun ouders.

We willen ons niet alleen op contextuele factoren richten (opvoeding in Hoofdstuk 2, familie geweld in Hoofdstuk 3). Daarom verzamelen we in Hoofdstuk 4 tweeling studies waarin de genetische invloeden op zelfcontrole wordt onderzocht. Tweelingstudies zijn een veelvoorkomend model binnen de gedragsgenetica om te begrijpen in hoeverre ons gedrag wordt verklaard door onze omgeving of door onze genen. Eeneiige tweelingen zijn genetisch vrijwel identiek, terwijl twee-eiige tweelingen gemiddeld 50% van hun genetische informatie delen (net als broers en zussen). In tweelingonderzoek wordt onderzocht in hoeverre eeneiige tweelingen meer op elkaar lijken dan twee-eiige tweelingen. Als de eeneiige-tweelinggelijkenis (de gelijkenis tussen een tweeling en zijn/haar tweelingbroer/zus) veel hoger is dan de twee-eiige tweeling gelijkenis, dan kan ervan uit worden gegaan dat genen een belangrijke rol spelen. Deze gelijkenis tussen tweelingen, en het verschil in gelijkenis tussen eeneiige en twee-eiige tweelingen, wordt vervolgens gebruikt om een erfelijkheidspercentage te berekenen. Uit onze meta-analyse van 31 studies blijkt dat de erfelijkheid van zelfcontrole 60% is. Deze erfelijkheidsschatting is hetzelfde voor jongens en meisjes, maar is hoger wanneer de zelfcontrole van het kind wordt gerapporteerd door de ouders dan wanneer de zelfcontrole alleen door het kind zelf wordt gerapporteerd.

Het erfelijkheidspercentage houdt in dat, op een populatieniveau, individuele verschillen in de mate van zelfcontrole tussen mensen voor 60% wordt verklaard door genetische verschillen tussen deze individuen. Een erfelijkheid van 60% betekent niet dat zelfcontrole niet te beïnvloeden is want een genetische schatting is geen deterministisch gegeven (Johnson, Turkheimer, Gottesman, & Bouchard, 2009). Het suggereert echter dat het voor sommige individuen gemakkelijker is om zelfcontrole uit te oefenen dan voor anderen, zelfs wanneer ze worden blootgesteld aan dezelfde interventie of omgeving, en dit wordt gedeeltelijk verklaard door iemands genetische profiel (Harold, Leve, & Sellers, 2017). Daarom moet men niet alleen rekening houden met de context (bijvoorbeeld opvoeding), maar ook met genetische verschillen als we de oorsprong van verschillen in de mate van zelfcontrole in de populatie willen begrijpen.

Samenspel van genen en omgeving

Individuele verschillen in zelfcontrole kunnen dus verklaard worden door de omgeving [Hoofdstuk 2, Hoofdstuk 3] en verschillen in het DNA [Hoofdstuk 4]. Historisch gezien werden deze twee perspectieven als twee uitersten gezien. De gedachte dat individuele verschillen zijn geworteld in de blootstelling aan onze omgeving (*nurture*) stond tegenover de gedachte dat individuele verschillen worden verklaard door onze biologie (*nature*) (Tucker-Drob & Bates, 2016). Inmiddels weten we dat genetische en omgevingsinvloeden elkaar niet wederzijds uitsluiten: de variatie in de populatie is het resultaat van de wisselwerking tussen de twee.

Het is uitermate belangrijk om deze wisselwerking mee te nemen in onderzoek. De samenhang van het gedrag van de ouder en het gedrag van het kind kan het resultaat zijn van een directioneel effect, maar het kan ook verklaard worden door gemeenschappelijke genetische factoren die zowel de gezinscontext als het gedrag van het kind beïnvloeden (genetische pleiotropie of genetische confounding, Pingault et al., 2018). Wanneer geen rekening gehouden wordt met genetische pleiotropie loopt een onderzoeker het risico om causale conclusies te trekken, terwijl er geen causaal verband is. Als zodanig is het belangrijk om de associaties tussen de familiecontext en zelfcontrole verder te testen [Hoofdstuk 2, Hoofdstuk 3], rekening houdend met omgevings- en genetische factoren [Hoofdstuk 4].

Voor mijn promotieproject maak ik gebruik van de data van het Nederlands Tweelingen Register (NTR), een grote longitudinale populatiestudie die al meer dan 25 jaar tweelingen en hun ouders en broertjes en zusjes in Nederland volgt. De rijkdom van deze data bied ons de mogelijkheid om een zelfcontroleschaal te maken en te valideren. In Hoofdstuk 5 testen we of de Achenbach System of Empirically Based Assessment (ASEBA) valide is om zelfcontrole te meten. We selecteren acht items, en testen de validiteit van de ASEBA-zelfcontroleschaal (ASCS) voor kinderen en adolescenten. De schaal heeft een goede interne consistentie en we zien significante associaties tussen (1) de ASCS en variabelen waar zelfcontrole theoretisch mee samenhangt (bijvoorbeeld educatieve prestaties, welzijn, middelengebruik), (2) verschillende informanten (bijvoorbeeld rapportage van de moeder, vader, leraar of het kind zelf) en (3) verschillende leeftijden (bijvoorbeeld van zeven tot zestien jaar). Onze erfelijkheidsschattingen komen overeen met de erfelijkheidsschattingen van andere studies (ongeveer 60%, zie Hoofdstuk 4). In Hoofdstuk 9 tonen we aan dat deze schaal ook valide is voor volwassenen.

De validiteit van de ASCS bij kinderen, tieners en volwassenen biedt een breed scala aan mogelijkheden voor onderzoekers om individuele verschillen in de mate van zelfcontrole te bestuderen. Ten eerste is de schaal betrouwbaar voor ouder-, leerkracht- en zelfrapportage, waardoor zelfcontrole in meerdere contexten kan worden gemeten. Ten tweede is weinig zelfcontrole bij kinderen een voorspeller van weinig zelfcontrole bij tieners en volwassenen. Daar komt bij dat zelfcontrole correleert met allerlei negatieve levensuitkomsten (gezondheidsproblemen, financiële problemen, kleiner sociaal netwerk, etc. Caspi et al., 2016; Duckworth, Tsukayama, & Kirby, 2013; Moffitt et al., 2011; Tsukayama, Toomey, Faith & Duckworth, 2010). Het gebruik van deze schaal kan mogelijk helpen bij het tijdig opsporen van kinderen die het risico lopen op de ontwikkeling van problemen die samenhangen met weinig zelfcontrole. Ten derde biedt de schaal mogelijkheden voor secundaire data-analyses. De ASEBA is een internationaal gebruikte schaal; meerdere grote longitudinale familiestudies hebben ASEBA-gegevens direct beschikbaar (bijv. EGDS, TCHAD, TRAILS, Leve et al., 2013; Lichtenstein, Tuvblad, Larsson, & Carlström, 2007; Ormel et al., 2012). Het aggregeren van bestaande internationale data zou veel toekomstig onderzoek kunnen faciliteren, zoals interculturele analyses (bijvoorbeeld het vergelijken van voorspellers voor de mate van zelfcontrole in verschillende landen), generatievergelijkingen (bijvoorbeeld het vergelijken van niveaus van zelfcontrole bij jongeren uit de jaren '80 en jongeren uit de jaren '00), snelle replicatie (bijvoorbeeld validering van de bevindingen in meerdere datasets) en ontwikkeling van statistische methoden (bijvoorbeeld gebruikmakend van de grote steekproefgroottes en replicatiesets).

De validatie van de ASCS in de NTR-data maakt het mogelijk om de relatie tussen de familiecontext en zelfcontrole te onderzoeken terwijl we rekening houden met zowel genetische als omgevingsinvloeden. In Hoofdstuk 6 onderzoeken we of sterke familiebanden en zelfcontrole causaal gerelateerd zijn. We vinden een significante, maar kleine associatie, wat suggereert dat adolescenten die sterkere familiebanden ervaren, meer zelfcontrole hebben. De aard van deze associatie wordt echter voornamelijk verklaard door gemeenschappelijke genetische factoren. In eeneiige tweelingparen (die 100% van hun genetica en familieomgeving delen) zien we dat de tweeling die sterkere familiebanden ervaart geen hogere zelfcontrole levels heeft dan de tweeling die mindere familiebanden ervoer. Dit houdt in dat, bij het interpreteren van de resultaten van de samenhang tussen familiebanden en zelfcontrole, men voorzichtig moet zijn bij de interpretatie van deze associaties als causaal.

In Hoofdstuk 7 onderzoeken we het verband tussen familieconflict en zelfcontrole tijdens de adolescentie, rekening houdend met zowel genetische als omgevingsfactoren. Hier testen we of de associatie tussen familieconflict en zelfcontrole wordt verklaard door: 1) gemeenschappelijke genetische factoren, 2) een bi-directionele invloed tussen familieconflict en zelfcontrole, 3) een directionele associatie van familieconflict die verminderde zelfcontrole veroorzaakt, of 4) een directionele associatie met verminderde zelfcontrole die familieconflicten verklaart. Door het zogenoemde 'Direction of Causation' model toe te passen (Duffy & Martin, 1994; Heath et al., 1993), tonen we aan dat er een directioneel effect bestaat waarbij familieconflict voor verminderde zelfcontrole zorgt, zelfs wanneer we rekening houden met genetische pleiotropie. Dus terwijl de associatie tussen positieve familieband en zelfcontrole waarschijnlijk niet causaal van aard is, lijkt het verband tussen familieconflict en zelfcontrole wél causaal te zijn. Lage zelfcontrole kan dus mede veroorzaakt worden door geweld in het gezin. Onderzoekers en behandelaars zouden hier rekening mee moeten houden wanneer ze verdere zelfcontroleproblemen willen begrijpen en voorkomen.

In tweelingstudies wordt onderzocht in hoeverre verschillen in de populatie worden verklaard door omgevings- of genetische factoren. Het maakt het bijvoorbeeld mogelijk om de algehele erfelijkheid van een eigenschap te onderzoeken (zie Hoofdstuk 3, 4), of om de rol van de omgeving op een uitkomst te onderzoeken terwijl we rekening houden met genetische pleiotropie (zie Hoofdstuk 6, 7). De tweelingmodellen gebruiken echter geen informatie over de specifieke genetische varianten die invloed hebben op ons gedrag. Binnen de moleculaire genetica wordt hier wel aandacht aan besteed. Zo wordt er met behulp van genoombrede associatiestudies (Genome-wide association studies, GWAS) gezocht naar specifieke genetische codes die individuele verschillen verklaren.

Het doel van Hoofdstuk 8 is om moleculaire genetische informatie te gebruiken om de wisselwerking tussen genen en omgeving verder te onderzoeken. In overeenstemming met de diathese-stresstheorie wordt verondersteld dat iemands genetische risico interacteert met omgevingsstressoren (ook wel gen omgevingsinteractie G x E genoemd, Kendler & Eaves, 1986; Monroe & Simons, 1991). Tot dusverre werd deze hypothese vooral getest met kandidaatgenstudies. Deze studies houden echter geen rekening met de polygenetische aard van complexe eigenschappen zoals zelfcontrole (Dick et al., 2015; Duncan & Keller, 2011). Daarom onderzoeken we de diathese-stresstheorie voor zelfcontrole, maar dan met polygenetische scores in plaats van kandidaatgeninformatie. We

zien dat een polygenetisch risico en stressvolle gebeurtenissen verminderde zelfcontrole voorspellen (weliswaar met een klein effect). We vinden echter geen significante interactie, dus we zien niet dat iemands genetische risico op verminderde zelfcontrole verder tot uiting komt wanneer iemand blootgesteld was geweest aan bepaalde omgevingsstressoren.

De diathese-stresshypothese is aantrekkelijk omdat het een theoretisch kader biedt dat verklaart hoe *nature* en *nurture* samen ons gedrag bepalen. Empirisch blijft het echter een uitdaging om statistisch bewijs voor deze hypothese te vinden. Er is meer onderzoek nodig om te begrijpen hoe we de interactie tussen genen en omgeving in kaart kunnen brengen. Misschien moeten we onze focus verleggen en ons meer richten op de correlatie tussen genen en omgeving (de associatie tussen ons genotype en de omgeving die we ervaren) in plaats van op de interactie tussen genen en omgevingen. De mate van zelfcontrole (gedeeltelijk erfelijk) van een persoon beïnvloedt namelijk de manier waarop we onze omgeving waarnemen, en hoe we onze omgevingen selecteren, wat resulteert in een associatie tussen onze genetische aanleg en onze omgeving.

Conclusie

De titel van dit proefschrift bestaat uit twee componenten. 'Out of Control' verwijst naar de individuele verschillen in de mate van zelfcontrole die we in de populatie zien, waarbij sommigen meer problemen hebben dan anderen om zelfcontrole te tonen. 'Oorzaken van individuele verschillen in zelfcontrole' verwijst naar het doel om te begrijpen welke factoren deze individuele verschillen verklaren. Met behulp van meta-analyses en tweelingmodellen tonen we aan dat zowel omgevingsfactoren (familiecontext) als genetische factoren (schatting van de erfelijkheid van 60%) een belangrijke rol spelen. Bovendien moeten onderzoekers en behandelaars zich ervan bewust zijn dat verminderde zelfcontrole kan voortvloeien uit de blootstelling aan geweld en conflicten in het gezin. In dit proefschrift benadrukken we dat het onderzoek naar de wisselwerking tussen genen en omgeving zeer complex is, maar noodzakelijk om de oorzaken van verschillen tussen mensen in hun mate van zelfcontrole te begrijpen. Zowel *nature* als *nurture* verklaren wie we zijn, maar achterhalen hoe deze wisselwerking precies werkt blijft een uitdaging die toekomstig onderzoek zal aangaan.

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Curriculum Vitae

ABOUT THE AUTHOR

Yayouk Eva Willems (1990, Nijmegen) obtained a Bachelor's degree in psychology and sociology at Amsterdam University College (2012, *summa cum laude*), and a Research Master's degree in clinical and developmental psychopathology at the Vrije Universiteit Amsterdam (2015, *cum laude*). During her studies, she worked as a project assistant for the publication of the "Handboek Systeemtherapie" (2011-2014), spent an exchange semester at the Boğaziçi Üniversitesi in Turkey (2012), and wrote her master thesis at the Early Growth and Development (EGDS) lab at the Pennsylvania State University in the U.S.A (2015). After graduating, she was a board member of the Amsterdam University College Alumni Association (2012-2013), and a board member of "Kinderen uit de Knel" (2015-2019, a program targeting families involved in high-conflict divorces).

In 2015, Yayouk was granted the research talent fund by the Netherlands Organisation for Scientific Research (NWO onderzoekstalent beurs). Her four-year PhD project was centered around individual differences in self-control, and supervised by prof. dr. Catrin Finkenauer (Universiteit Utrecht) and prof. dr. Meike Bartels (Vrije Universiteit Amsterdam). She presented her work at international conferences, including the International Workshop on Statistical Genetic Methods for Human Complex Traits (Boulder 2016), the European Conference on Developmental Psychology (ECDP, Utrecht 2017), the European Association of Development Psychology (EARA, Ghent 2018), and the Behavior Genetics Associations (BGA, Boston 2018, Stockholm 2019). In 2018, she spent one month at the Education University of Hong Kong to collaborate with dr. Jianbin Li. Together with prof. dr. Catrin Finkenauer, she edited a special issue on family violence for the International Journal of Environmental Research and Public Health, and wrote a chapter for the Organization for Economic Co-operation and Development (OECD) on adolescent relationships in the 21st century.

During her PhD project, Yayouk supervised bachelor and master students with their theses and coordinated the tutors of the bachelor's course "Interplay of Genes and Environment". Additionally, Yayouk aimed to communicate her research to a wider audience. She became part of the department's social media team, participated in the "Weekend van de Wetenschap" to promote science to young children, talked about twin research on national television, and participated in the "Science Battle" theater show. She very much enjoyed the collaboration with her colleagues at the department of Biological Psychology at the Vrije Universiteit Amsterdam, where she organized multiple department activities. Currently, Yayouk is exploring life outside of academia.

PUBLICATIONS

Finkenauer, C., Büyükcan-Tetik, A., Schoemaker, K., **Willems, Y. E.**, Bartels, M., & Baumeister, R. F. (2018). Examining the role of self-regulatory strength in family violence. In D. de Ridder, M. Adriaanse, & K. Fujita (Eds.), *The Routledge international handbook of self-control in health and wellbeing* (pp. 340–352). London, UK: Routledge.

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Li, J. B.*, **Willems, Y.E.***, Stok, F. M., Deković, M., Bartels, M., & Finkenauer, C. (2019). Parenting Self-Control across Early to Late Adolescence: A Three-level Meta-analysis. *Perspectives on Psychological Science*, *14*(6).

Willems, Y. E., Laceulle, O. M., Bartels, M., & Finkenauer, C. (revise & resubmit). Investigating the association between family connectedness and self-control in adolescence in a genetically sensitive design. Preprint available at PsyArxiv: 10.31234/osf.io/yxjdp.

Willems, Y. E., Hottenga, J. J., Ligthart, L., Willemsen, G., Boomsma, D. I., Finkenauer, C., & Bartels, M. (submitted). Stressful life events and self-control: Testing gene x environment interaction with a polygenic approach. Preprint available at PsyArxiv: 10.31234/osf.io/jhbqd.

Li, J. B., Bi, S., **Willems, Y. E.**, Finkenauer, C. (submitted). School discipline and self-control from preschoolers to high school students: A meta-analysis. Preprint available at PsyArxiv: 10.31234/osf.io/8nk2x.

Hendriks, A. M., **Willems, Y. E.**, Genomic SEM Consortium, Nivard, M. G. (in preparation). Anxiety and depression: Using genomic structural equation modeling to understand their co-morbidity.

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Grants / Awards

Erasmusbeurs	2012	€ 1.000
A.S.C. Academy	2014	€ 1.000
Stichting Bekker – la Bastide Fonds	2014	€ 500
Stichting de Fundatie van de Vrijvrouwe van Renswoude	2015	€ 1.000
Vreedefonds	2015	€ 2.000
Behavioural Genetics Association Travel Award + Registration Fee	2015	€ 337
NWO Research Talent	2015	€ 219.170
FGB Talent Fund	2016	€ 1.300
APH Personal Medicine Travel Grant	2018	€ 1.000
Nominated for the Lindon Eaves Poster Price	2016	
Winner Science Battle NWO Synergy Conference	2019	

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